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Original Articles

A POSSIBILITY FOR PHYSIOLOGIC ORTHODONTIC MOVEMENT

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THEORETICAL PART

INTRODUCTION

THE histologic investigation on tissue changes due to artificial forces was initiated by Sandstedt,[36] in 1904, in experimenting on dogs. The author followed in 1911 with another contribution, using monkeys as experimental animals.[25] Since then, further research work has been done by other experimenters and by the author himself. Though it would seem that the subject had been exhausted and nothing new could be added, some new findings justify this contribution. The experiments reported here were done to test previous statements[28, 29] that intermittent forces give results different from those produced by continuous pressure, and, further, that this reaction to gentle forces is not so severe and detrimental as that to strong ones.

GENERAL REMARK

In the present emergency, the publisher is subject to restrictions, and illustrations have to be cut down to the utmost. Therefore, in several cases, the author will not show photographs of certain details if they can be clearly described, or if they can be demonstrated in other pictures.

FINDINGS

Mature monkeys *(Macaca rhesus)* were used in three experiments, each comprising four incisors. For each, a plain arch (precious metal, 0.03 inch thick) was inserted into buccal tubes soldered to caps on the molars and premolars and tied to the banded incisors with steel ligatures. Coil springs

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furnished the continuous force in the first and third experiments, while the elasticity of the labial arch alone provided the intermittent pressure in the second.

The coil springs of stainless steel wire 0.010 inch in diameter were 1 cm. long with the individual coils separated from each other by 1 mm. They were compressed between the mesial ends of the tubes and spurs on the arches to different lengths; in one experiment, to one-half their original length, in the other, to two-thirds. The force necessary for such a compression amounted, in the first case, to 180 grams, in the latter case, to 120 grams. But as two springs, one on each side, were used, the pressure exerted on the teeth amounted to 360 and 240 grams, respectively. It must not be forgotten that every spring gradually becomes fatigued, depending on the material as well as on the duration of the compression. For instance, the coil spring, 1 cm. long, loaded for five days with 120 grams, returned to only 8 mm. in length. So the pressure actually delivered can only be estimated.

In the first series, four lower incisors were moved labially by the continuous force of coil springs compressed to half their length and acting through a plain labial arch fastened to them by ligatures (360 grams). The individual teeth were moved for one, two, three, and four days.

In Series 2, four upper incisors were ligated to a plain labial arch standing 1 mm. away from them. The ligatures were either tightened or replaced every other day. After six days of movement, they were permitted to relapse for one, two, three, and four days.

In the third series, four other upper incisors were moved as in Series 1; the coil springs were compressed to two-thirds of their length (240 grams) and were active through twenty to twenty-three days, when the teeth were permitted to relapse for one, two, three, and four days.

SERIES 1 (COIL SPRINGS; 360 GRAMS; ONE TO FOUR DAYS)

In Fig. 1 (Spec. 313), we see in higher magnification the labial crest of a lower lateral incisor that was moved in this direction for one day with coil springs of 10 mm. length, that were compressed to half of their original length, thereby exerting in the beginning a pressure of approximately 360 grams. The periodontal membrane (*Pd,* Fig. 1) is compressed to 0.09 mm., while on the lingual traction side it is seven times greater (0.6 mm.). The number of cells and their staining properties are reduced and some kind of hyalinization or fibrination is noticeable (*h,* Fig. 1). Just below the compressed area the cells assume again their normal appearance (*a,* Fig. 1). The bone surface facing the periodontium is smooth and aplastic (*Bs,* Fig. 1), while on the other hand quite numerous "secondary osteoclasts" are found on the periosteal side (*Occ,* Fig. 1), although pressure was active only twenty-four hours. They are removing the crest, the principal obstacle to movement. The term "secondary osteoclasts" will be explained later under *Discussion.* The osteocytes (bone corpuscles) are nearly normal although several are pyknotic; a few bone lacunae are empty. The cementoid seam and the cementoblasts have almost disappeared in the compression area. They reappear distinctly beyond this region at C_1. As to the condition on the apical pressure side, nothing can be said. This part of the

specimen was cut away in securing it. As in the other specimens of this series, numerous hemorrhages and lively osteoclastic activity on the bone surface were found on the traction sides at the crest as well as in the apical region, occasionally also in the neighboring marrow spaces. In the discussion we will try to give an explanation for this unusual picture.

Fig. 1.—(Spec. 313.) Lower incisor; higher magnification of labial crest. *D*, Dentine; *C*, cementum; C_1, cementum seam, distinct; *Pd*, periodontal membrane; *ac*, alveolar crest; *Po*, periosteum; *Occ*, osteoclasts; *Bs*, aplastic periodontal bone surface; *h*, hyalinized tissue of the periodontal membrane; *a*, normal periodontal membrane; arrow, direction of movement.

Fig. 2 (Spec. 310) shows in higher magnification the labial crest of a lower lateral incisor that was moved for two days. The periodontal membrane (*Pd*, Fig. 2) is entirely crushed for some distance and transformed into a disorganized mass of tissue without any cells. Approximately two-thirds of the crushed area is shown in the picture. Sometimes thrombosed vessels are found; the width has been reduced to 0.08 mm. as compared to 0.82 mm. on the traction side, ten times as much. The bone surface (*Bs*, Fig. 2) facing the periodontium is aplastic; while on the periosteal side, osteoclastic activity is already well under way at two places (*Occ*, Fig. 2). The condition of the osteocytes in the pressure area is impaired; many are pyknotic, stain faintly, and some bone lacunae are empty. Within the compression area the cementum also is aplastic. The cementoid seam and the cementoblasts in the region of the greatest pressure have completely disappeared (*pr*, Fig. 2). On the lingual side, due to the

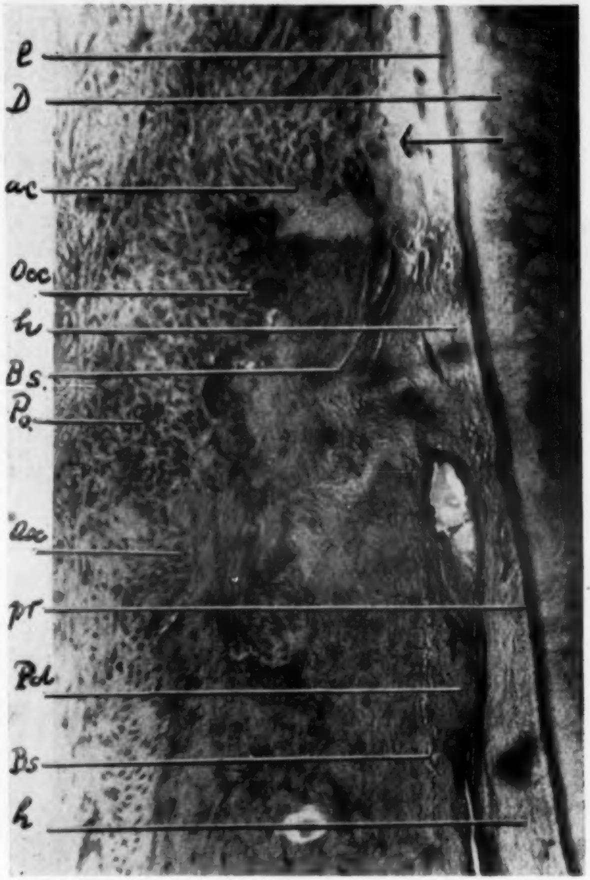

Fig. 2.

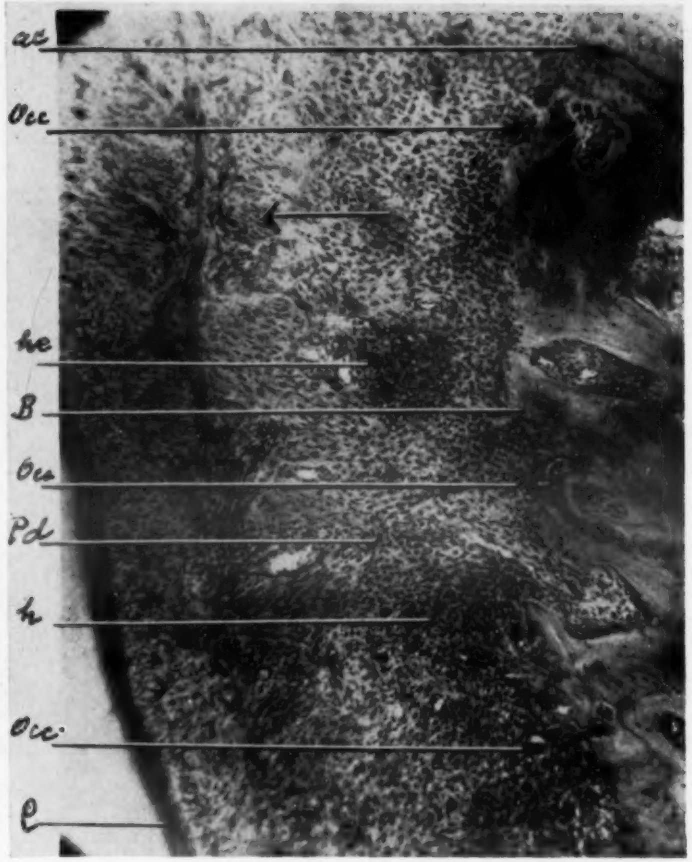

Fig. 3.

Fig. 2.—(Spec. 310.) Lower incisor; higher magnification of labial crest. *D*, dentine; *C*, cementum and cementoid seam; *Pd*, periodontal membrane; *ac*, alveolar crest; *Po*, periosteum; *Bs*, aplastic periodontal bone surface; *h*, hyalinized tissue; *Occ*, osteoclasts; *pr*, aplastic cementum; arrow, direction of movement.

Fig. 3.—(Spec. 310.) Lower incisor; higher magnification of lingual crest (traction side). *C*, Cementum; *B*, bone; *ac*, alveolar crest; *Pd*, periodontal membrane; *h*, *he*, hemorrhages; *Occ*, osteoclasts; arrow, direction of movement.

strong traction, we find again the unusual picture (Fig. 3). Most of the fibers are torn and only at their point of fixation to the cementum can their original course be followed for only a short distance. Near the bone surface they intersect each other in all directions. Numerous hemorrhages are found (*h, he,* Fig. 3) and the bone surface is covered with many osteoclasts (*Occ,* Fig. 3). Occasionally, they are also found in the marrow space. No indication of any osteoid formation is found. In the apical traction side we find the same pictures but not so pronounced; here also, hemorrhages and osteoclastic activity on the bone surface can be seen.

In Fig. 4, the hemorrhage (*he*) in Fig. 3 is reproduced in high magnification.

The deviation of the apex is quite considerable, brought about by the fulcrum established at the crest. The amount of the deviation can be judged by the difference in the width of the periodontal membrane; on the lingual pressure side it measures 0.09 mm., on the labial traction side 0.5 mm, six times as much. Cementum resorptions are not present.

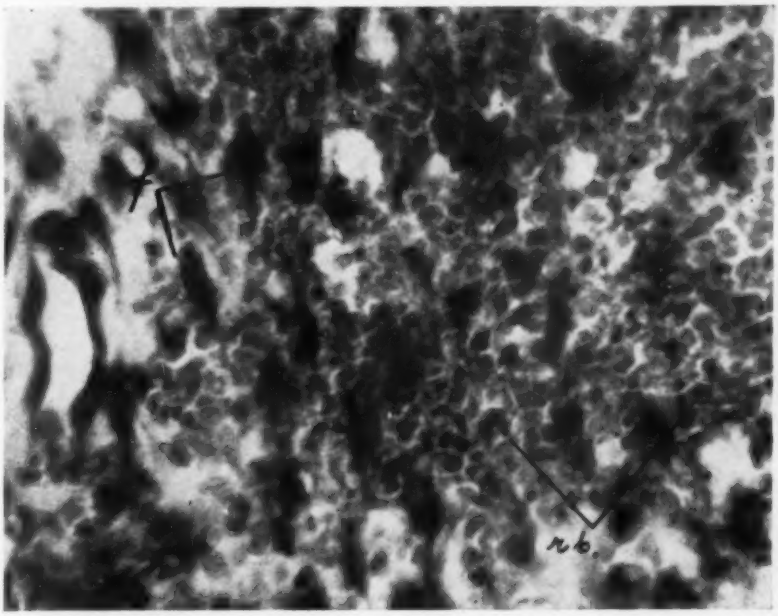

Fig. 4.—High magnification of *he,* hemorrhage in Fig. 3. *f,* Fibroblast, *rb,* red blood corpuscles.

Fig. 5 (Spec. 312) represents in higher magnification the labial crest of the lower central incisor moved labially for three days. The findings are similar to those in the first two specimens of one and two days' movement, only more pronounced. The periodontal membrane (*Pd,* Fig. 5) is crushed and partly hyalinized (*h,* Fig. 5). Very few cells and, occasionally, thrombosed vessels can be seen. The crushed area is not as extended as in the previous specimens nor is the difference in the labial and lingual widths of the periodontal membrane so pronounced. These circumstances justify the assumption that the ligature must have become loose within the last day, thus decreasing the force transmitted. The periodontal width measures labially 0.2 mm., lingually 0.4 mm. The periodontal bone surface (*Bs,* Fig. 5) in the compression area is

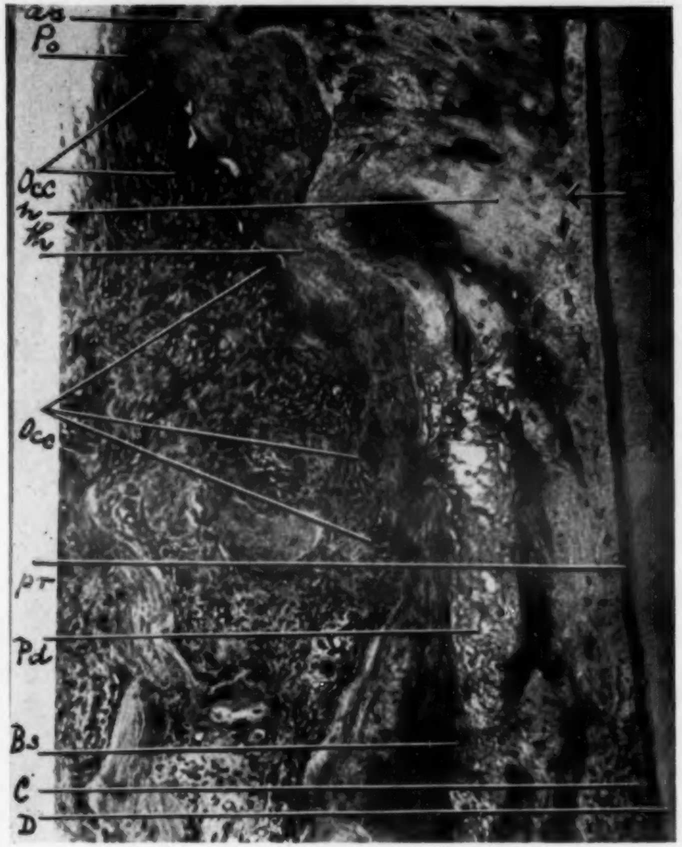

Fig. 5.

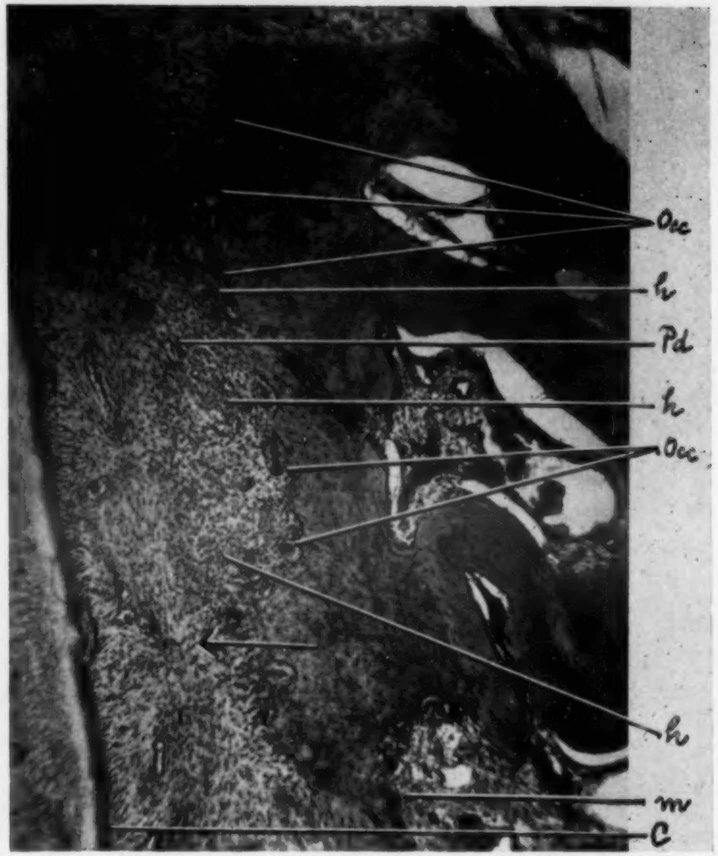

Fig. 6.

Fig. 5.—(Spec. 312.) Lower incisor; higher magnification of labial crest. *D*, Dentine; *C*, cementum; *Pd*, periodontal membrane; *Bs*, aplastic periodontal bone surface; *pr*, aplastic cementum; *Occ*, osteoclasts; *th*, place of greatest thinning of bone with bone corpuscles disappeared; *h*, hyalinized periodontal membrane; *Po*, periosteum; *ac*, alveolar crest; arrow, direction of movement.

Fig. 6.—(Spec. 312.) Higher magnification of lingual crest (traction side). *C*, Cementum; *Pd*, periodontal membrane; *Occ*, osteoclasts; *m*, osteoclasts in marrow space; *h*, hemorrhages; arrow, direction of primary movement.

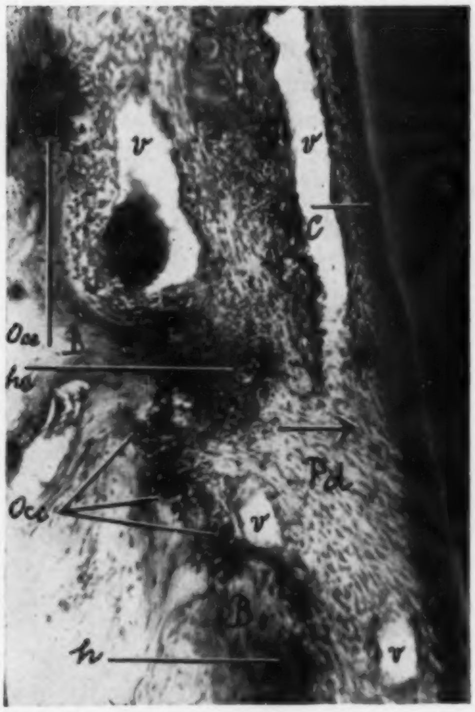

Fig. 7.—(Spec. 312.) Higher magnification of apical traction side. *C*, Cementum; *Pd*, periodontal membrane; *h*, *he*, hemorrhages, *Occ*, osteoclasts; *v*, vessels; arrow, direction of movement.

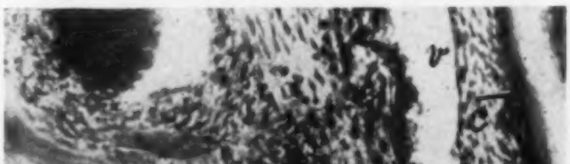

Fig. 8.—(Spec. 312.) Higher magnification of *he* in Fig. 7. *C*, Cementum; *B*, bone; *Pd*, periodontal membrane; *h*, *he*, hemorrhages; *Occ*, osteoclasts; *v*, vessels; arrow, direction of primary movement.

aplastic, while its periosteal surface becomes attacked by numerous secondary osteoclasts (*Occ*, Fig. 5) whose activity has reduced the thickness of the bone to 0.07 mm. Except in the region of the greatest thinning (*th*, Fig. 5), the osteocytes still present are, for the most part, normal. Some are pyknotic; only a few bone lacunae are empty.

The cementum in the compression area is devoid of the cementoid seam and the cementoblasts. Above and below this area, both reappear.

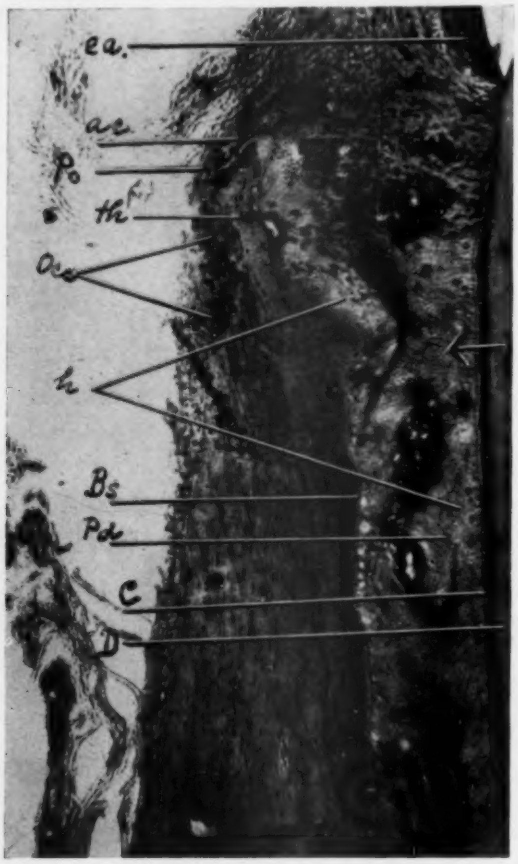

Fig. 9.—(Spec. 311.) Lower incisor; higher magnification of labial crest. *D*, Dentine; *C*, cementum with cementoid seam; *Pd*, periodontal membrane; *Bs*, aplastic periodontal bone surface; *Occ*, osteoclasts; *th*, place of greatest thinning of bone; *h*, hyalinized periodontal membrane; *ac*, alveolar crest; *Po*, periosteum; *ea*, epithelial attachment; arrow, direction of movement.

On the lingual traction side (Fig. 6), many fibers are torn and no functional structure is discernible. Many hemorrhages are present (*h*, Fig. 6) and the bone surface towards the periodontium is covered by numerous osteoclasts (*Occ*, Fig. 6). They are also found in quite a great number in some marrow spaces (*m*, Fig. 6). Though the force was in some way diminished, the damage was done within the first two days. Seldom is an indication of osteoid formation found.

On the apical traction side (Fig. 7), some fibers remained intact and caused in such places by their traction the formation of osteoid. But here also we find mostly ruptured vessels and hemorrhages as the result of too great a force (*he*, *h*, Fig. 7). The hemorrhage (*he*) in Fig. 7 is reproduced in middle high

magnification in Fig. 8 to show it and the many osteoclasts (*Occ*, Fig. 8) in its neighborhood simultaneously. This is not possible with high magnification.

Occasionally osteoclasts are found also in marrow spaces. The osteocytes are normal.

In Fig. 9 (Spec. 311) we see the last tooth in this series, the labial crest of a lower central incisor after four days of labial movement. The periodontal membrane is almost entirely hyalinized (*h*, Fig. 9) and measures 0.18 mm. as compared with 0.4 mm. on the lingual side. The periodontal bone surface (*Bs*, Fig. 9) is smooth and aplastic, while on the periosteal side secondary osteoclasts are at work (*Occ*, Fig. 9), having already reduced the thickness of the labial plate to 0.05 mm. (*th*, Fig. 9). The resorbed area is less extended than in Fig. 5. The osteocytes are affected only to a minor degree. The cementoid seam and the cementoblasts have not disappeared in the compression area (*C*, Fig. 9). In the compression area at the apex the same effects are produced as on the crest; crushed periodontal tissue, aplastic bone surface, deterioration of the osteocytes, undermining resorption.

On the lingual side as a result of the strong traction, we find the already familiar picture of hemorrhages and osteoclasts. On the crest, these effects are much more pronounced in the marrow spaces, while in the apical region, they are more noticeable on the surface.

SERIES 2 (LABIAL ARCH WITH LIGATURES)

This series (Spec. 252 to 255; Figs. 10 to 25) comprises four upper incisors that were moved for six days with ligatures tightened or renewed every other day. After six days of movement the teeth were allowed to relapse for one, two, three, and four days, respectively. To avoid unnecessary repetition it may be stated: the side towards which a tooth was originally moved is the primary pressure side (pr. pr. s.); the side from which it was originally moved is the primary traction side (pr. tr. s.). During the relapse the pr. pr. s. becomes the secondary traction side (s. tr. s.), and vice versa, and the primary traction side becomes the secondary pressure side (s. pr. s.).

The first photomicrograph in this series is shown in *Fig. 10 (Spec. 255)*. It is the outline picture of an upper lateral incisor, that, after six days of labial movement, was permitted to relapse for one day. The lingual rim of the still unformed and uncalcified root was bent somewhat labially (*b*, Fig. 10); it is shown in higher magnification at *br* in Fig. 13. In Fig. 11, we see the labial alveolar crest in higher magnification (*a* from Fig. 10). The periodontal membrane is crushed and homogeneous (*h*, Fig. 11); all cells have disappeared; some hemorrhages are present (*he*, Fig. 11). The crushed area is quite extended. The periodontal bone surface is aplastic and smooth (*Bs*, Fig. 11); but on the periosteal surface secondary osteoclasts are at work to remove the crest (*Occ*, Fig. 11), similar to Figs. 1, 2, 5, and 9. Besides these secondary osteoclasts we find others, *primary osteoclasts*, in quite a great number just above the aplastic area on the periodontal surface in an area of diminished pressure. This part is not shown. These primary osteoclasts on the now s. tr.s. still persist though twenty-four hours have elapsed since the force was discontinued; there was no reason any more for their presence.

Above the periosteal resorption, well-developed osteophytes (*Oph*, Fig. 11) more pronounced in neighboring slides, are visible. The beginning of such a formation can be seen in Fig. 16, *Oph*. Both phenomena, the resorption and the osteophytes, are not equally well developed in the same slide. If one is well advanced, the other is poor.

Fig. 10.—(Spec. 255.) Outline picture; upper incisor. *a*, Labial alveolar crest; *b*, lingual rim of the root end which is not yet fully formed and calcified is bent toward the labial; *c*, crushed periodontal membrane; arrow, direction of primary movement.

Within the aplastic bone area, all osteocytes have disappeared in the periodontal half of the buccal plate; the lacunae are empty as in Fig. 24 or 28. They are separated by a cementing line (*cl*, Fig. 11 and *l*, Fig. 28) from the periosteal half, where the osteocytes are present and well stained.

The cementum surface is aplastic (*C*, Fig. 11).

On the lingual traction side (Fig. 12) taken from a neighboring slide, we see the effect of a strong but not too strong force; the bone follows the traction with individual spicules because not too many fibers were broken. In contrast with the findings in the first series, we do not find any hemorrhages or osteoclastic activity since the causes for their presence, too strong forces, are not present.

Osteoclasts cannot yet be seen on the osteoid surface after twenty-four hours of increased pressure caused by the relapse.

On account of the relapse of one day's duration, the periodontal width at the crest on the s. tr. s. is now somewhat greater (0.3 mm.) than on the lingual

s. pr. s. (0.2 mm.). This difference of 0.1 mm. is somewhat smaller at the apex; the width here at the apex on the lingual s. tr. s. amounts to 0.15 mm. in comparison to 0.07 mm. on the labial s.pr. s., or a difference of 0.08 mm. Though these measurements prove that the relapse occurred relatively slowly, nevertheless we find several pressure areas on the lingual s. pr. s. with the tissues crushed, homogeneous, and without cells. In such places the width amounts to 0.05 mm.

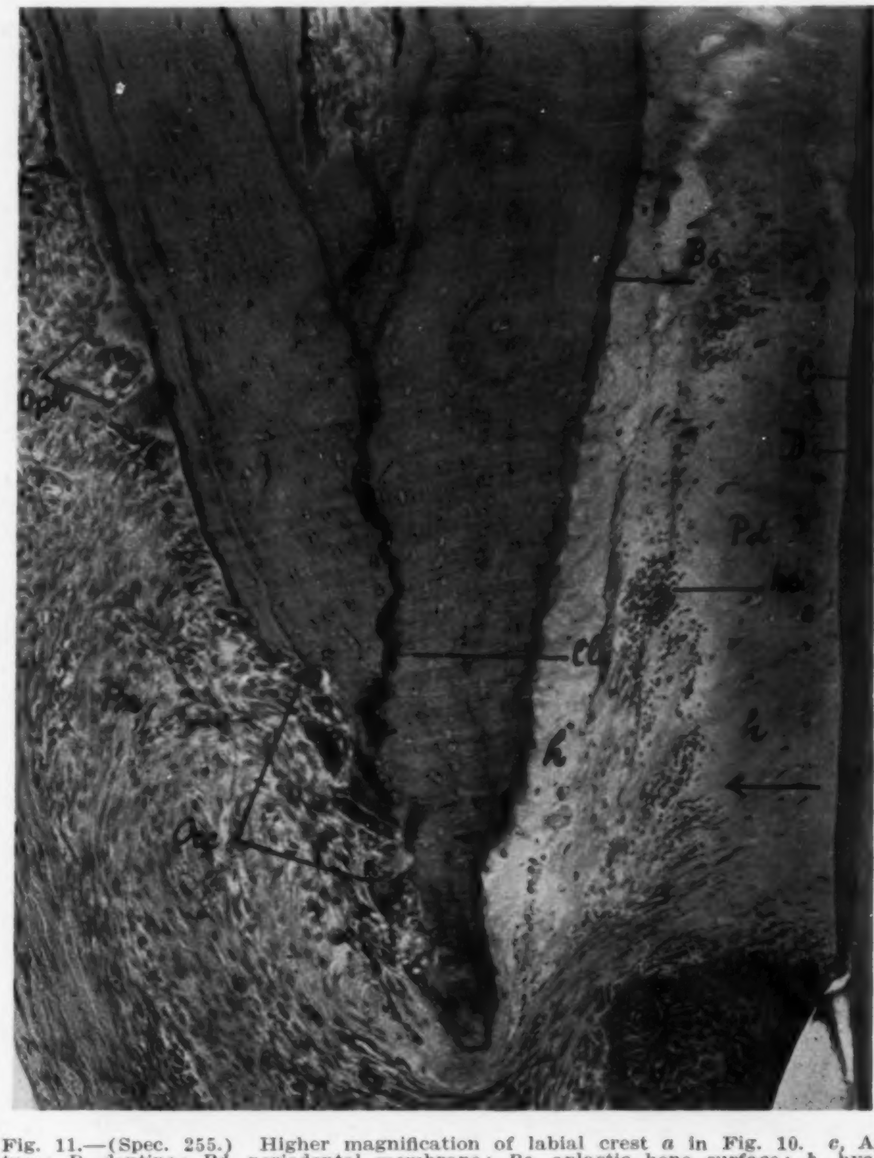

Fig. 11.—(Spec. 255.) Higher magnification of labial crest *a* in Fig. 10. *c*, Aplastic cementum; *D*, dentine; *Pd*, periodontal membrane; *Bs*, aplastic bone surface; *h*, hyalinized periodontal membrane; *he*, hemorrhage; *Occ*, osteoclasts on the periosteal bone surface; *cl*, cementing line; *Pr*, periosteum; arrow, direction of primary movement.

The osteocytes in these areas of short duration of the compression (one day) are intact. Also, osteoclasts have not yet developed here.

On the apical pr. pr. s. (Fig. 13, a higher magnification of *c* in Fig. 10), the periodontal tissue (*Pd*, Fig. 13) has nearly entirely disappeared. Some tiny fibers only and homogenized tissue at the borders are visible. Undermining

resorption from the marrow spaces (*Occ*, Fig. 13) and the neighboring intact periodontium (*Ocl*, Fig. 13) is under way. The bone surface (*Bs*, Fig. 13) is aplastic; all lacunae in the bone near the periodontium are empty. The osteocytes have disappeared entirely (*el*, Fig. 13, and Fig. 14, a high magnification of *bc* in Fig. 13). The condition caused by crushing is at its peak. Deeper, near the marrow space, the bone cells are again present and well stained (*bc*, Fig. 14).

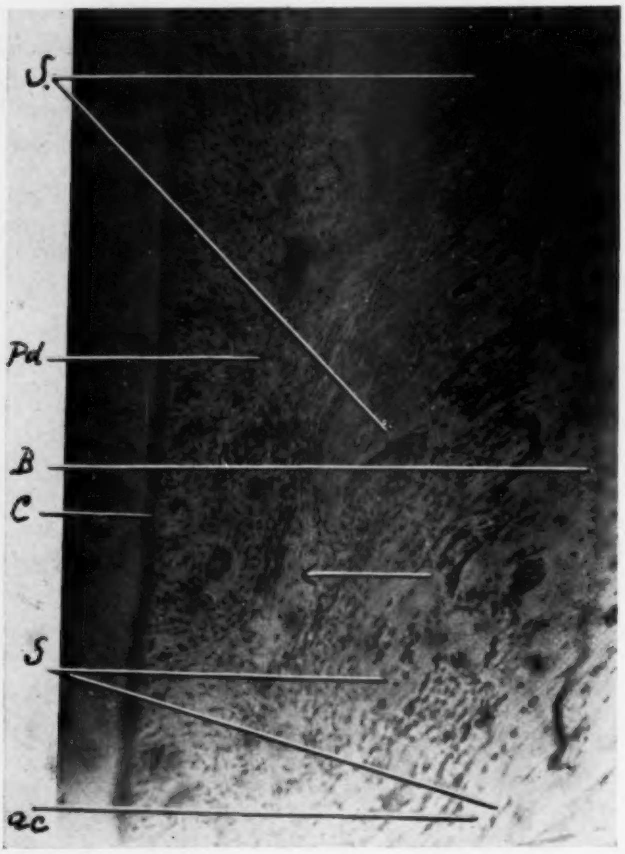

Fig. 12.—(Spec. 255.) Higher magnification of the lingual crest (traction side) *d* from Fig. 10. *C*, Cementum; *B*, bone; *S*, elongated osteoid spicules following the traction; *Pd*, periodontal membrane; *ac*, alveolar crest; arrow, direction of movement.

These cells are more remote from the lifeless periodontal membrane (*Pd*, Fig. 14) and furthermore their nourishment is furnished from the nearby marrow space (*ms*, Fig. 14). In still higher magnification, this is shown in Fig. 15. In the superficial cementum layers adjacent to the periodontium, most of the cementum corpuscles are also pyknotic or have disappeared (*eC*, Fig. 14, high magnification of *C* in Fig. 13), while in the deeper older layers they maintained their normal appearance (*r*, Fig. 14). In the compression area both the cementoid and the cementoblasts have disappeared (*Cs*, Fig. 14).

In the whole series there was no indication of any cementum resorption.

In the beginning and towards the end of the series, when we approach the approximal sides, the periodontal membrane becomes gradually wider on account of the curving root surface. For this reason and because the force is

transmitted here at a more or less acute angle (see sketch, Fig. 37), the pressure becomes diminished. We see, therefore, that the periodontal tissue has maintained its vitality and ability to react. The bone is removed from the periodontal side all along the area of increased pressure by primary osteoclasts only (*Oc*, Fig. 16), without any signs of undermining resorption. The bone is removed physiologically. The location of these primary osteoclasts corresponds to the location of the osteoclasts (tertiary) in Figs. 3 and 6, though they have quite a different significance, as will be explained in the discussion.

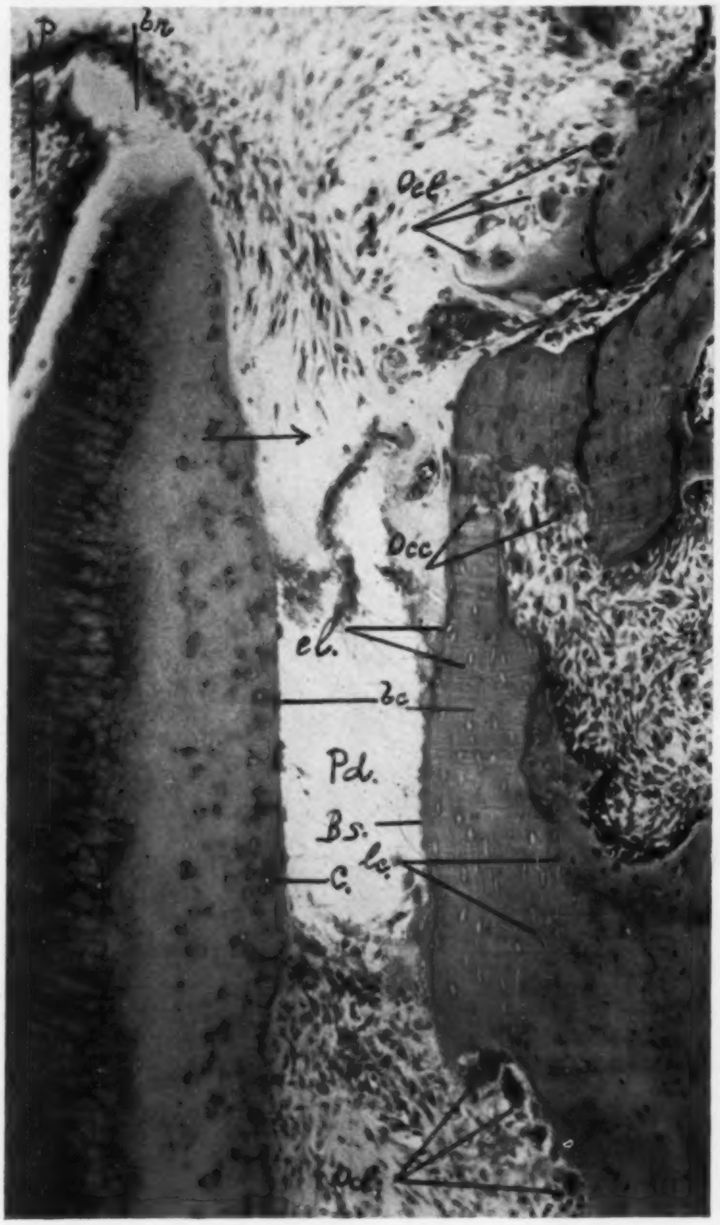

Fig. 13.—(Spec. 255.) Higher magnification of *c* in Fig. 10. *P*, pulp; *br*, bent root end; *Pd*, crushed periodontal membrane; *bs*, aplastic bone surface; *Occ*, osteoclasts in marrow space; *Ocl*, osteoclasts in the neighborhood of the crushed periodontal membrane; *C*, cementum, aplastic surface; *el*, empty bone lacunae; *lc*, living osteocytes; arrow, direction of primary movement.

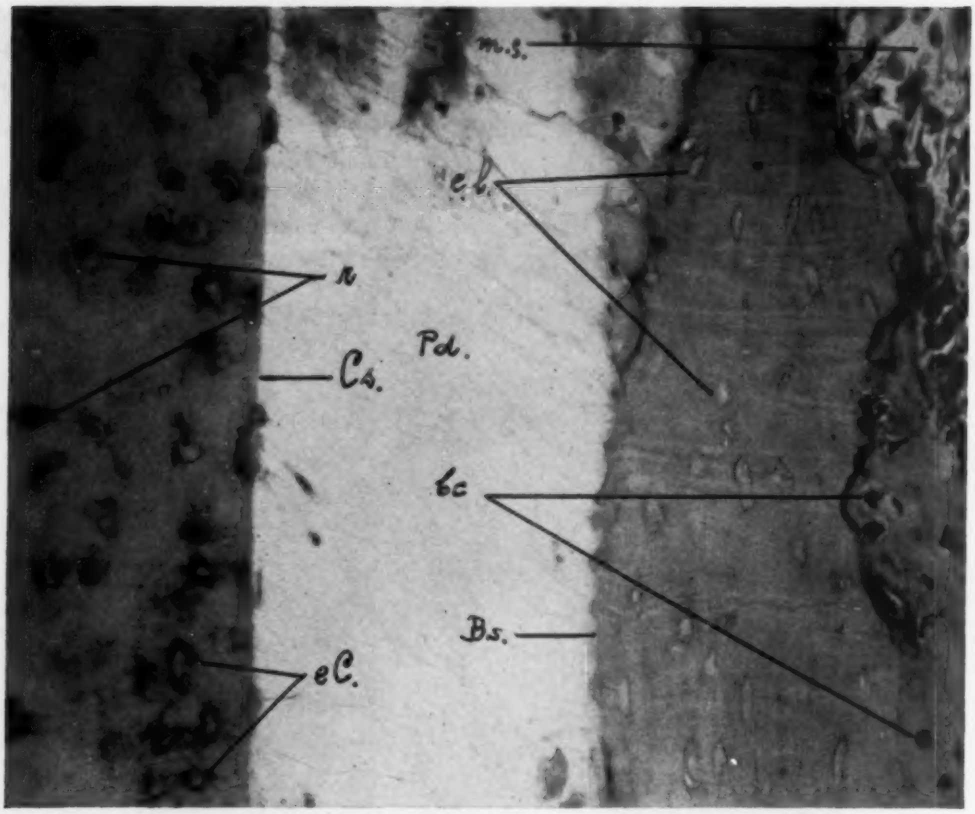

Fig. 14.—(Spec. 255.) High magnification of *bc* in Fig. 13. *Pd*, crushed periodontal membrane with only some tissue fibers left; *Bs*, aplastic bone surface; *Cs*, aplastic cementum surface; *ms*, marrow space; *el*, empty bone lacunae; *eC*, empty cementum lacunae; *bc*, osteocytes present; *r*, living cementum corpuscles.

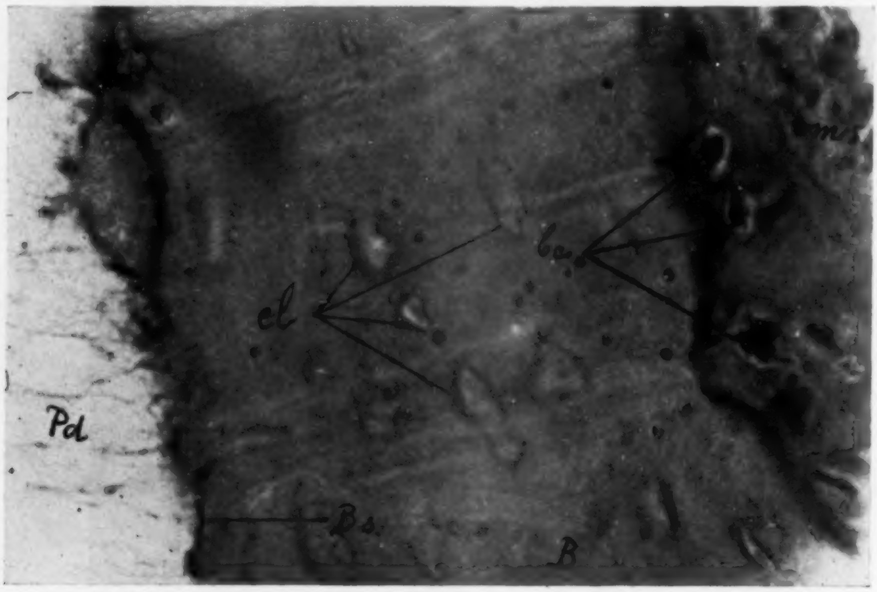

Fig. 15.—(Spec. 255.) Still higher magnification of *el* in Fig. 14. *Pd*, remnants of periodontal membrane. *B*, Bone; *el*, empty bone lacunae; *bc*, osteocytes in the lacunae; *ms*, marrow space; *Bs*, aplastic bone surface.

The beginning of osteophytic bone formation, to maintain the bone equilibrium, is seen at ***Oph,*** Fig. 16. At the crest proper, a tiny bit of bone is still left (*ac,* Fig. 16), covered with five osteoclasts. In drawing a line from the tip of ***ac,*** parallel to the bone surface, one gets an idea of the amount of bone already removed by the resorption. The osteocytes are not affected; they are normal.

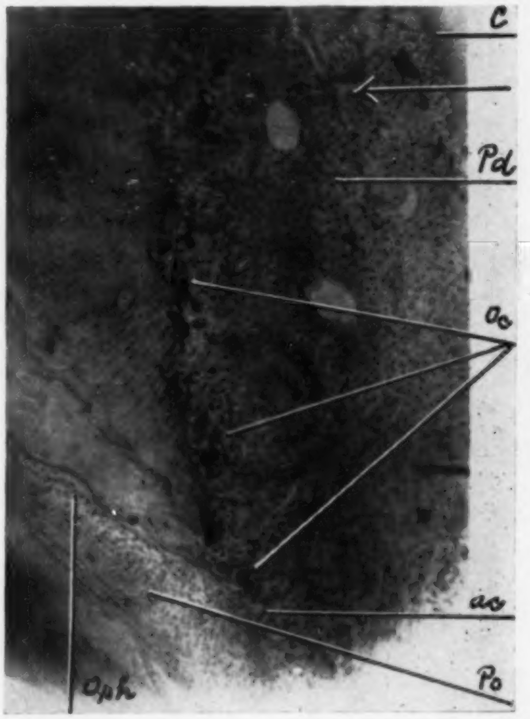

Fig. 16.—(Spec. 255.) Alveolar labial crest in the region of diminished pressure; lively primary osteoclastic activity on the periodontal bone surface. *Oc,* osteoclasts; a quite thin remnant of bone (*ac*) is still left at the crest; *Pd,* periodontal membrane; *C,* cementum; *Po,* periosteum; *Oph,* starting osteophytic bone formation on periosteal bone surface; arrow, direction of primary movement.

In Fig. 17 (Spec. 254) we see the outline picture of an upper central incisor after six days of labial movement and two days of relapse. The periodontal membrane in the two pressure areas at the crest and apex does not show any crushing of the tissues; only the number of cells seems to be somewhat diminshed (***Pd,*** Figs. 18 and 20). The ligature probably became loose and the pressure must have been of minor degree or so low that it did not even stimulate the formation of osteoclasts, or at least, not of multinucleated osteoclasts. Two days after the starting of relapse, or even earlier, the formation of osteoid starts. This osteoid, covered with osteoblasts, is seen in Fig. 18, ***Bs,*** a higher magnification of ***a*** in Fig. 17. On the periosteal side, osteoclastic activty is beginning (***Occ,*** Fig. 18) though the osteocytes are not affected to a great degree. They are somewhat pyknotic and only a few empty lacunae are found. Just a little above the crest, not reproduced here, primary osteoclasts still are found on the periodontal bone surface forty-eight hours after force discontinuation. Here the periodontal space must have been somewhat narrower so that the initial insignificant pressure was able to incite the formation of these osteoclasts.

The periodontal surface of the bone at the crest on the lingual s. pr. s. shows the most favorable reaction to the slight original traction; an even layer of osteoid apposition without elongated spicules (*Ost,* Fig. 19, higher magnification of *c,* Fig. 17). This reaction, too, has to be considered the result of the ligature loosening. Also, the relapse movement must have occurred relatively slowly for we do not find any crushed areas on this lingual s. pr. s. But, on the other hand, no osteoclasts of either sort have yet developed on the osteoid formed during the primary movement, though the increased pressure due to the relapse has been present for forty-eight hours.

Fig. 17.

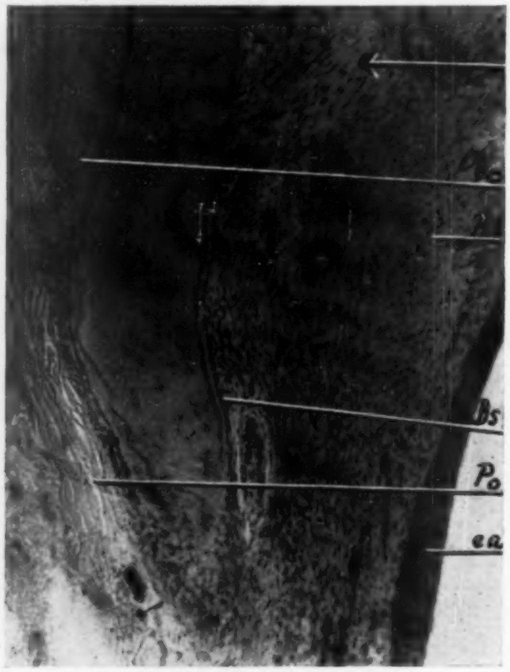

Fig. 18.

Fig. 17.—(Spec. 254.) Outline picture; upper incisor. *a,* Labial alveolar crest; *b,* primary apical pressure area; arrow, direction of the primary movement.

Fig. 18.—(Spec. 254.) Higher magnification of *a* in Fig. 17. *Pd,* periodontal membrane; *Po,* Periosteum; *ea,* epithelial attachment; *Bs,* periodontal bone surface with osteoid seam; *Occ,* osteoclasts on periosteal bone surface; arrow, direction of primary movement.

In Fig. 20, we see in higher magnification the lingual apical pr. pr. s. corresponding to *b,* Fig. 17 and taken from a neighboring slide. Here, also, we find a seemingly normal periodontal membrane, but in contrast to the conditions at the crest, the cells responded to the light pressure. We find a physiologic bone reaction—primary osteoclasts on the periodontal bone surface only (*Oc,* Fig. 20). These osteoclasts are still active two days after force discontinuation. In the adjoining marrow space, instead of finding the signs of undermining resorption—the common picture after application of stronger forces (*Occ,* Fig. 13)—we find compensatory osteophytic bone formation (*Oph,* Fig. 20). The individual bone spicules, instead of being weakened, maintain at least their

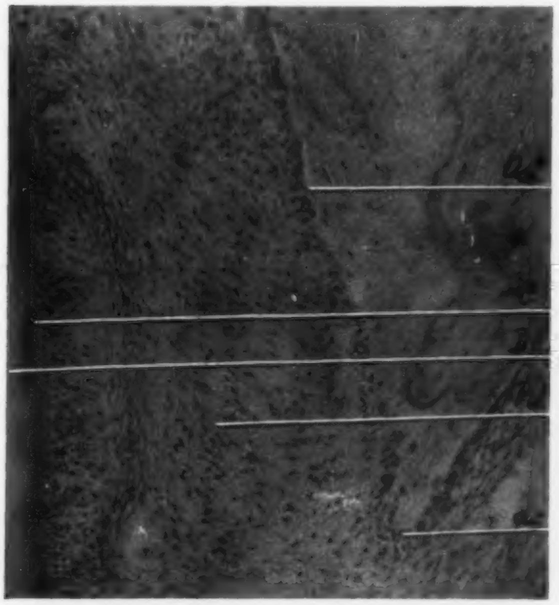

Fig. 19.—(Spec. 254.) Higher magnification of lingual alveolar crest *c* Fig. 17. *C*, cementum; *D*, dentine; *Pd*, periodontal membrane; *ac*, alveolar crest; *ost*, osteoid seam with osteoblasts.

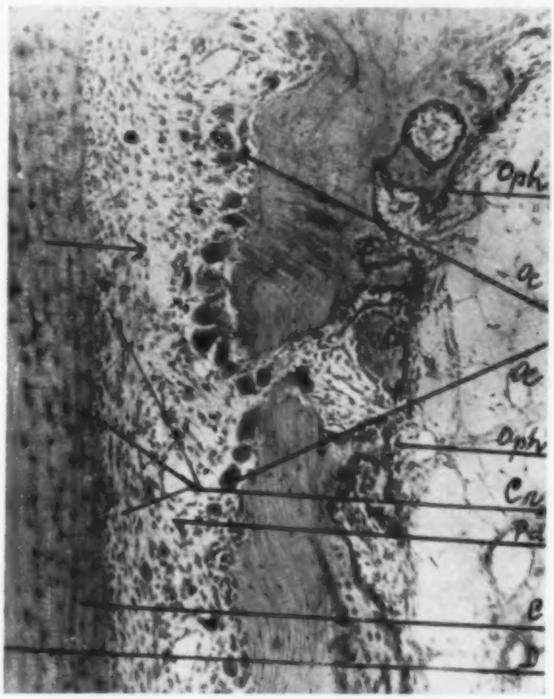

Fig. 20.—(Spec. 254.) Apical primary pressure side in higher magnification, *b* from Fig. 17. *C*, Cementum; *D*, dentine; *Pd*, periodontal membrane; *Oc*, osteoclasts; *Cr*, cementoclasts in shallow resorptions; *Oph*, osteophytic bone formation in marrow space; arrow, direction of primary movement.

original strength, perhaps become stronger temporarily, thus strengthening the bony support. Besides these primary osteoclasts we find, also, the cementoclasts, after two days of force elimination, still at work in the shallow cementum resorptions (*Cr*, Fig. 20).

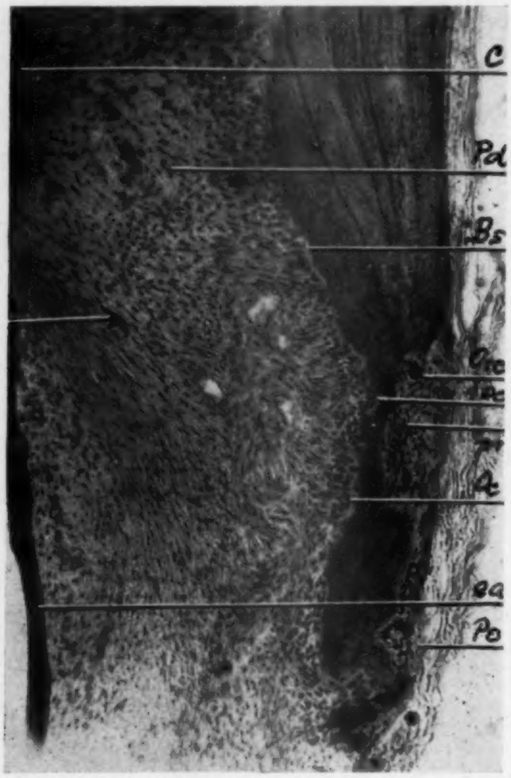

Fig. 21.—(Spec. 253.) Upper incisor; labial alveolar crest in higher magnification. *C*, Cementum with cementoid seam and cementoblasts; *ea*, epithelial attachment; *Pd*, periodontal membrane; *Bs*, scalloped bone surface with some osteoclasts (*Oc*) still present; *Po*, periosteum; *pr*, periosteal resorption with some secondary osteoclasts (*Occ*) still present; arrow, direction of primary movement.

In Fig. 21 (Spec. 253) we see in higher magnification the labial crest of an upper central incisor that was allowed to relapse for three days after having been moved labially for six days with ligatures. On the scalloped periodontal bone surface of the pr. pr. s., primary osteoclasts (*Oc*, Fig. 21) are still found, though the pressure has been eliminated for three days. But simultaneously the bone becomes resorbed by secondary osteoclasts from the periosteal side reducing its thickness there to 0.04 mm. (*pr*, Fig. 21). Some of these osteoclasts are still present (*Occ*, Fig. 21). The osteocytes in some places, as in the thin strip of remaining bone, are either pyknotic or missing. In other places in the compression area, they seem not to be affected. The periodontal tissue is altered only to a minor degree; the cells do not stain properly. It must be assumed that, in this case also, the ligatures became loose towards the end of the experiment, reducing thereby the force, or that the tissues have already recovered to some degree during the three days of force discontinuation. On account of the normal conditions and the less impaired nourishment, the cementoid seam and the cementoblasts, too, did not disappear or they have reappeared.

They are clearly discernible (*C*, Fig. 21). On the lingual s. pr. s. at the crest, some areas of crushed tissue are present. The periodontal membrane measures 0.07 mm. as compared to 0.3 mm. on the labial s. tr. s., four times as much. By the increased pressure of three days' duration, osteoclasts have developed on the surface of the osteoid bone laid down during the primary movement, in places where the tissues were not crushed or when the pressure did not rise above a certain level.

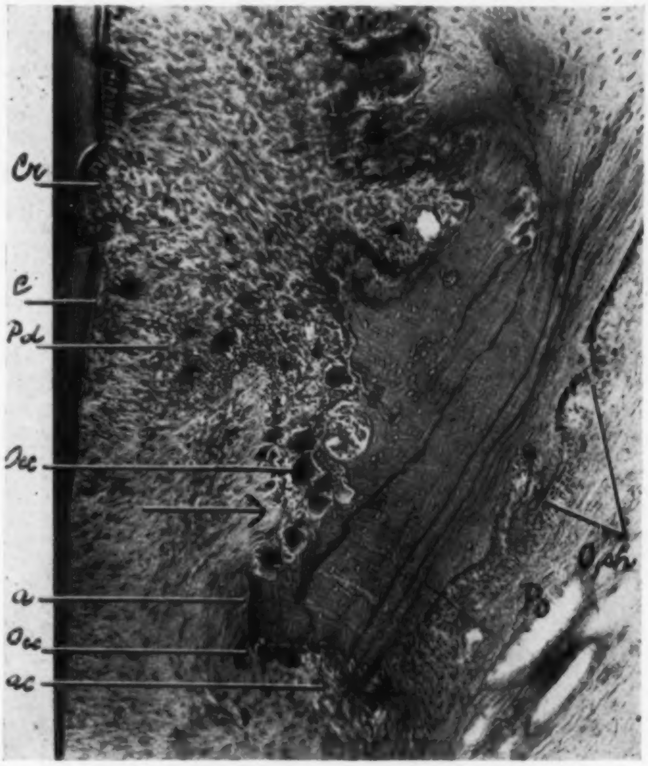

Fig. 22.—(Spec. 252.) Upper incisor; labial alveolar crest in higher magnification. *C*, Normal cementum; *Pd*, periodontal membrane; *Cr*, cementum resorption; *ac*, alveolar crest; *Occ*, secondary osteoclasts; *Oph*, osteophytic bone formation; *Po*, periosteum; *a*, aplastic bone not yet removed by undermining resorption (*Occ*); arrow, direction of original movement.

In Fig. 22 (Spec. 252) we see in higher magnification the labial crest of an upper lateral incisor moved in this direction for six days with ligatures and permitted to relapse for four days. On this tooth, the ligatures seem to have remained tight, for all the findings point to this assumption. On the pr. pr. s. near the crest, the periodontal membrane (*Pd*, Fig. 22) has recovered somewhat during the four days of the relapse, for the cells are reappearing in greater number. That the force applied was too great can be concluded from the fact that undermining resorption is at work (*Occ*, Fig. 22). Part of the aplastic bone is still not resorbed (*a*, Fig. 22). No resorption on the periosteal side can be seen, but it may have been present on the bone that already has disappeared at the crest. Just above the resorbed crest area, we find starting osteophytic bone formation (*Oph*, Fig. 22). The persistence of primary osteoclasts above the crest in the area of diminished pressure could not be demonstrated in this specimen after four days of pressure discontinuation. But we will see them

later in another specimen (302, Fig. 35, *Oc*) after the same elapse of time. Near the crest on the lingual s. pr. s., multinucleated primary osteoclasts (*Oc*, Fig. 23) are present on the bone surface that during the primary movement was covered with a strip of osteoid bone. It took four days before this osteoid became attacked by osteoclasts. These osteoclasts developed because the pressure during the relapse did not rise above a certain level. The relapse must have taken place at a low rate, for the width of the periodontal membrane buccally and lingually is nearly the same; at the crest it measures 0.3 mm. on the buccal as against 0.2 mm. on the lingual s. pr. s. On places like *n* in Fig. 23, the width of the periodontal membrane is reduced by the projection of two spicules towards the tooth. The tissue is hyalinized; undermining resorption has started (*Occ*, Fig. 23). The width at *n* measures 0.0183 mm., at *Pd* 0.13 mm.; a difference of only 0.1 mm. is responsible for the difference in the reaction.

Fig. 23.—(Spec. 252.) Upper incisor; lingual s.pr.s. just above the crest. *D*, dentine; *Pd*, periodontal membrane; *B*, bone; *Oc*, primary osteoclasts; *Occ*, secondary osteoclasts; *n*, crushed periodontal membrane; arrow, direction of relapse movement.

To come back to the labial alveolar crest (Fig. 22), we find the disintegration of the osteocytes again at its peak. Most of the lacunae are empty across the whole thickness of the labial plate, as can be seen in Fig. 22. An area corresponding approximately to *a* in Fig. 22 and taken from a neighboring slide, is shown in Fig. 24 in high magnification. We see the empty bone lacunae (*el*, Fig. 24), while, in the neighborhood of a marrow space (*ms*, Fig. 24), the osteocytes are found to be present again (*bc*, Fig. 24). A detail of this illustration in still higher magnification is shown in Fig. 25 (*x* of Fig. 24).

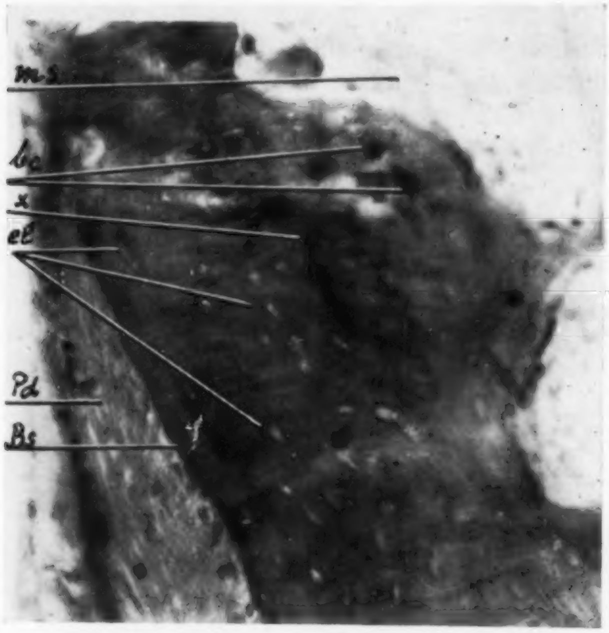

Fig. 24.—(Spec. 252.) High magnification of area *a* in Fig. 22, taken from a neighboring slide. *Pd*, Periodontal membrane; *Bs*, aplastic bone surface; *el*, empty bone lacunae; *bc*, osteocytes present in the neighborhood of marrow space (*ms.*) providing nourishment.

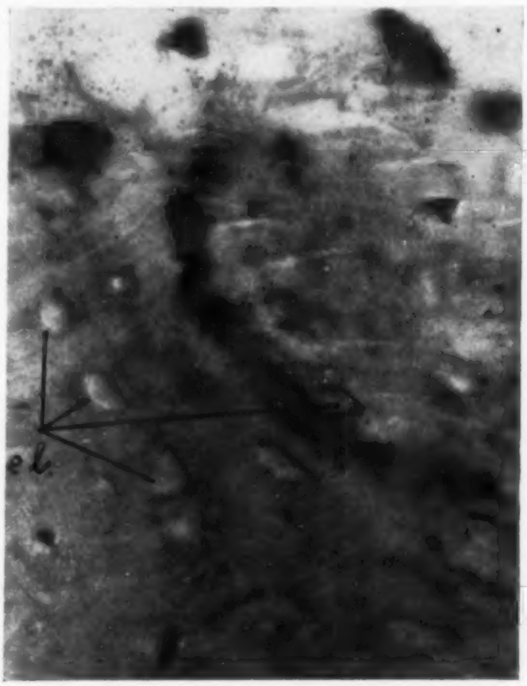

Fig. 25.—(Spec. 252.) Still higher magnification of *x* in Fig. 24. *el*, Empty bone lacunae.

The osteocytes on the pr. tr. s. are normal. In Fig. 22, *Cr*, we see also a cementum resorption with a few cementoclasts still present after four days of force discontinuation. The resorption is not very deep or extended; it can be followed in only twenty-four slides.

Fig. 26.—(Spec. 304.) Outline picture; upper incisor. *a*, Labial alveolar crest; *P*, apex not yet closed.

SERIES 3 (COIL SPRINGS; 240 GRAMS; 20 TO 23 DAYS)

The third series comprises four upper incisors that were moved labially for twenty to twenty-three days with coil springs of 1 cm. length compressed to two-thirds their original length, thereby exerting in the beginning a pressure of approximately 2 times 120, or 240 grams. They were adjusted in the usual way and readjusted after thirteen days; then they continued to work for ten more days. The teeth were allowed to relapse for one, two, three, and four days, respectively, before the animal was sacrificed.

Fig. 26 (Spec. 304) shows the outline picture of an upper central incisor that was moved labially for twenty-three days by the continuous force of the coil spring and was permitted to relapse for one day. The periodontal membrane, compressed and crushed in the first stages of the original movement, became restored to nearly normal. The pressure became reduced by various causes: by bone resorption, by the decrease of the force in the progress of movement as the spring extended itself gradually toward its original length, and by

the fatigue of the spring. It had subsided entirely during the twenty-four hours of the relapse.

In Fig. 27, a higher magnification of *a* in Fig. 26, we see, on the labial crest, undermining resorption of the periodontal (*oc*) as well as the periosteal side (*occ*) of the bone. In both places, secondary osteoclasts are still at work as in *oc*, Fig. 27. But while some of the aplastic bone is still present (*ap*, Fig. 27), we find on the remaining part, primary osteoclasts (*ocl*, Fig. 27) forming the superficial Howship's lacunae. By the osteoclastic activity, the alveolar

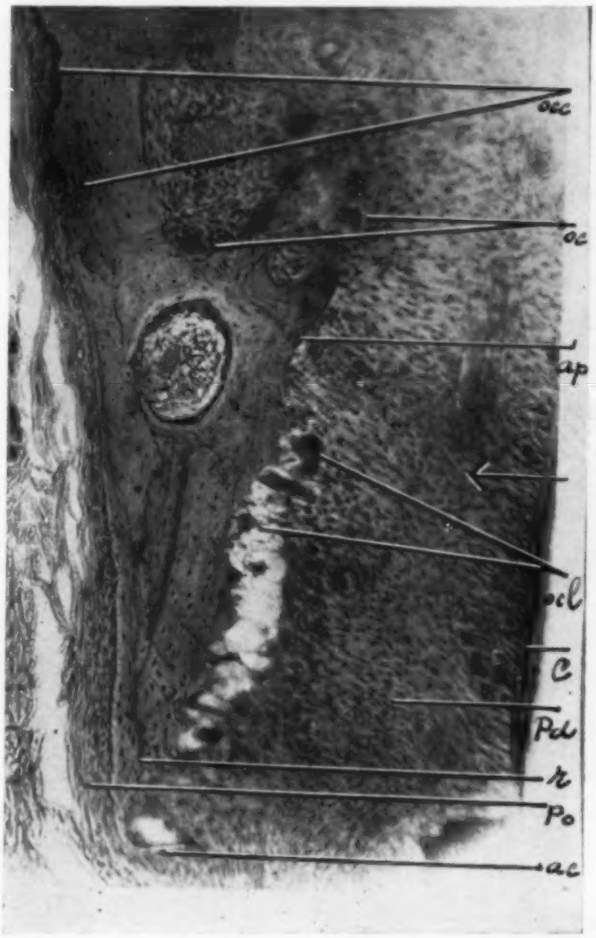

Fig. 27.—(Spec. 304.) Higher magnification of *a* in Fig. 26. *ac*, Alveolar crest entirely hollowed out; *Pd*, periodontal membrane; *Po*, periosteum; *oc*, secondary osteoclasts on periodontal side of bone; *occ*, secondary osteoclasts on periosteal side; *ocl*, primary osteoclasts in Howship's lacunae; *C*, cementum; *ap*, remnants of aplastic bone; *r*, cementing line separating the periodontal from the periosteal part of bone; arrow, direction of primary movement.

crest (*ac*, Fig. 27) was hollowed out entirely, leaving just a tiny spicule not supported by any osteophytes. Here also, as in Specimen 255, primary osteoclasts are present (*ocl*, Fig. 27) twenty-four hours after force discontinuation when their presence would seem to be no longer required.

In the pressure area in nearly the whole series, the osteocytes have disappeared entirely in the inner two-thirds of the bone adjacent to the periodontium.

In Fig. 28, a high magnification of *r* in Fig. 27, taken from the next neighboring slide, we see the empty lacunae (*el*) in the periodontal two-thirds and a cementing line (*l*, Fig. 28) separating it from the periostal third, where the lacunae contain normal well-stained cells (*bc*, Fig. 28) on account of the better nourishment of this part provided by the periosteum (*Po*, Fig. 28). Not all empty lacunae nor those containing still living cells can be seen sharply at the same time. To see many more, one must focus higher or deeper with the objective.

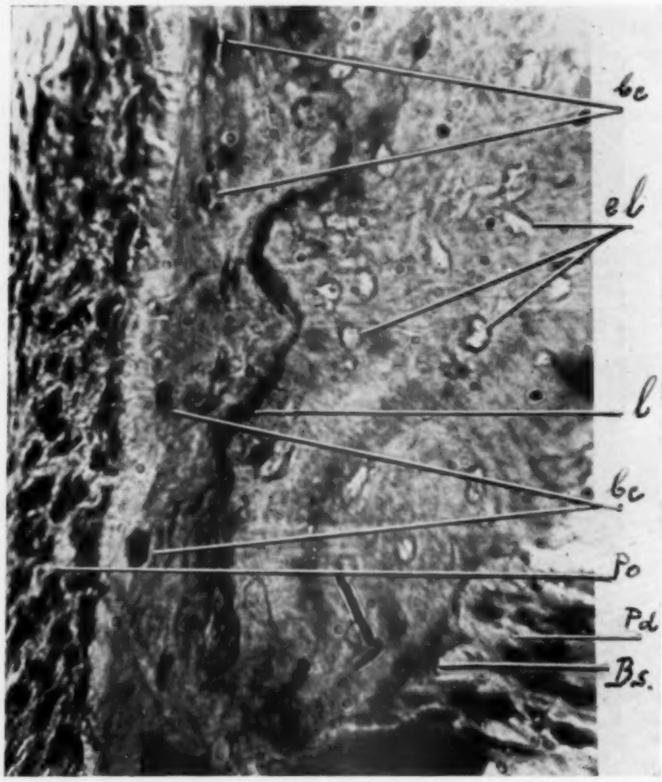

Fig. 28.—(Spec. 304.) High magnification of *r* in Fig. 27 taken from a neighboring slide. *Pd*, Periodontal membrane; *Bs*, scalloped bone surface; *Po*, periosteum; *l*, cementing line, separating the periodontal from the periosteal part of bone; in the periodontal part the bone lacunae are empty (*el*), while in the periosteal part the osteocytes are preserved (*bc*).

In the apical pressure area, the osteocytes were found normal. Maybe the affected bone has been eliminated in the twenty-three days that the experiment lasted. In the apical traction area we find, in many specimens, the results of the primary traction still present: many hemorrhages and numerous osteoclasts on the bone surface similar to Figs. 3 and 6.

Even after twenty-three days, the osteophytic bone formation, usually quickly and well developed in monkeys under the application of light forces, is here very poor; just a vestige can be found in several slides.

The width of the periodontal membrane at the crest on the labial s. tr. s. is 0.36 mm. as compared to 0.18 mm. on the lingual s. pr. s., where in some places the periodontal membrane is compressed to such a degree that it can no longer be considered normal. The number of cells is reduced and their staining is poor.

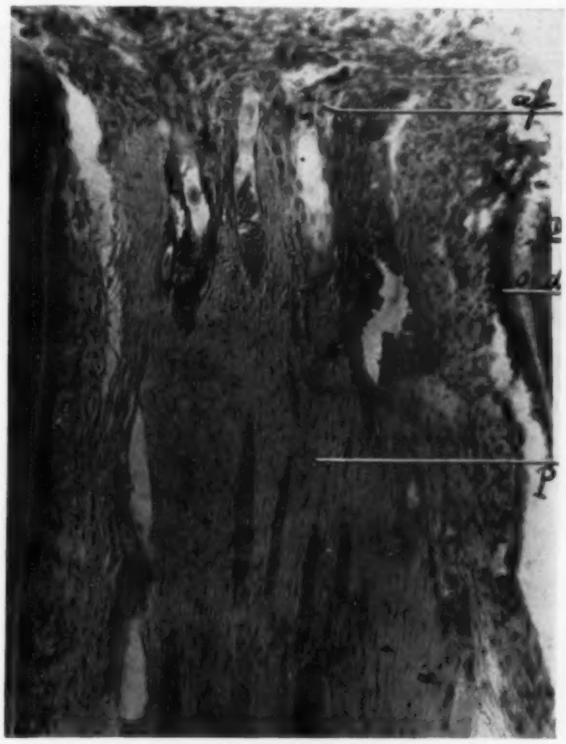

Fig. 29.—(Spec. 304.) High magnification of *P* in Fig. 26. *af*, Apical foramen; *P*, transformed pulp tissue; *D*, dentine; *Od* odontoblasts; twenty-three days' movement with coil spring and one day relapse.

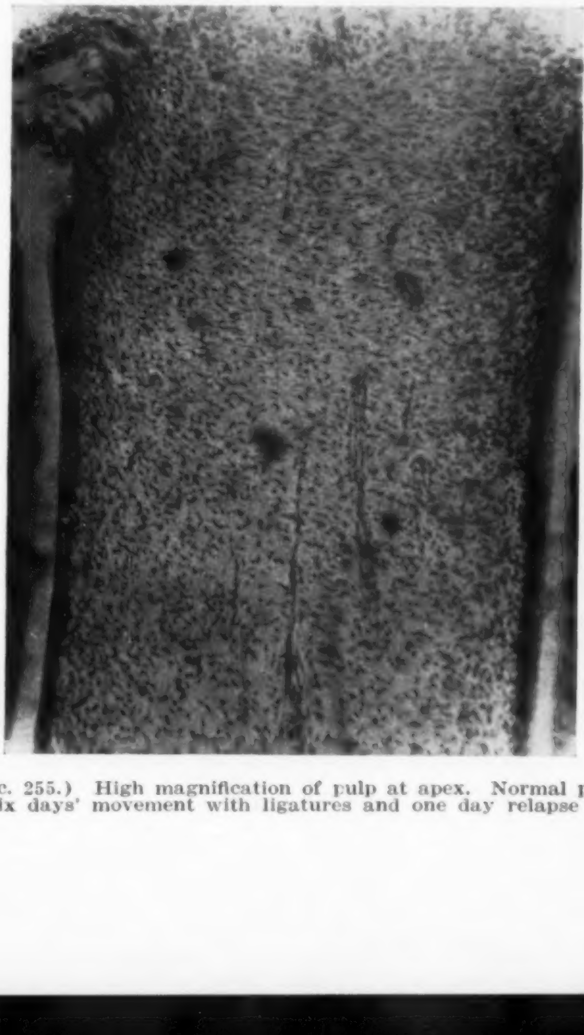

Fig. 30.—(Spec. 255.) High magnification of pulp at apex. Normal pulp tissue and normal odontoblasts; six days' movement with ligatures and one day relapse corresponding to *P* in Fig. 13.

The effect of the primary traction is clearly to be seen (wide osteoid borders) but they have not yet been attacked by osteoclasts after twenty-four hours of increased pressure caused by the relapse.

At the apex, the width of the periodontal membrane on the labial s. pr. s. is 0.1 mm., five times less than on the lingual s. tr. s. (0.5 mm.). Many primary osteoclasts are still present. After the removal of the original bone by undermining resorption, the pressure in the periodontal membrane became decreased and this decreased pressure is responsible for the formation of these primary osteoclasts that are still present twenty-four hours after force discontinuation.

Fig. 31.—(Spec. 305.) Outline picture; upper incisor. *a*, Alveolar labial crest; *b*, compression area during the relapse movement.

The great difference in the width of the periodontal membrane at the apex (five times) indicates its great excursion during the original and the relapse movement. The reaction of the pulp cannot fail to be present. After twenty-three days we see the beginning of metaplastic changes, that can be followed, though in a decreasing degree, from the apex upward, to the middle third of the root.

In Fig. 29, a higher magnification of *P* in Fig. 26, we can clearly follow the considerable reduction in the number of cells and the loss of their characteristic starlike shape. They assume more the character of a connective tissue structure. The odontoblasts are normal in structure and arrangement. For comparison with a pulp that after only six days of more gentle movement has not yet undergone pathologic changes, see Fig. 30 taken from Specimen 255 (*P*, Fig. 13). The cementum is intact.

In Fig. 31 (Spec. 305) we see the outline picture of an upper right lateral incisor that was moved labially for twenty-two days by the same coil spring as the tooth shown in Fig. 26 and was permitted to relapse for two days. As the crest has been resorbed (Fig. 32, a higher magnification of *a* in Fig. 31) its original height cannot be estimated any more. The aplastic bone facing the periodontium has for the greatest part disappeared, as has the crest itself. Where some aplastic bone is still present (*ab*, Fig. 32), the secondary osteoclasts (*Occ*, Fig. 32) are still at work removing it. No osteoclastic activity whatsoever is found at the periosteal smooth bone surface. The still remaining but decreased pressure caused the appearance of primary osteoclasts (*Oc*, Fig. 32); they are present two days after force discontinuation.

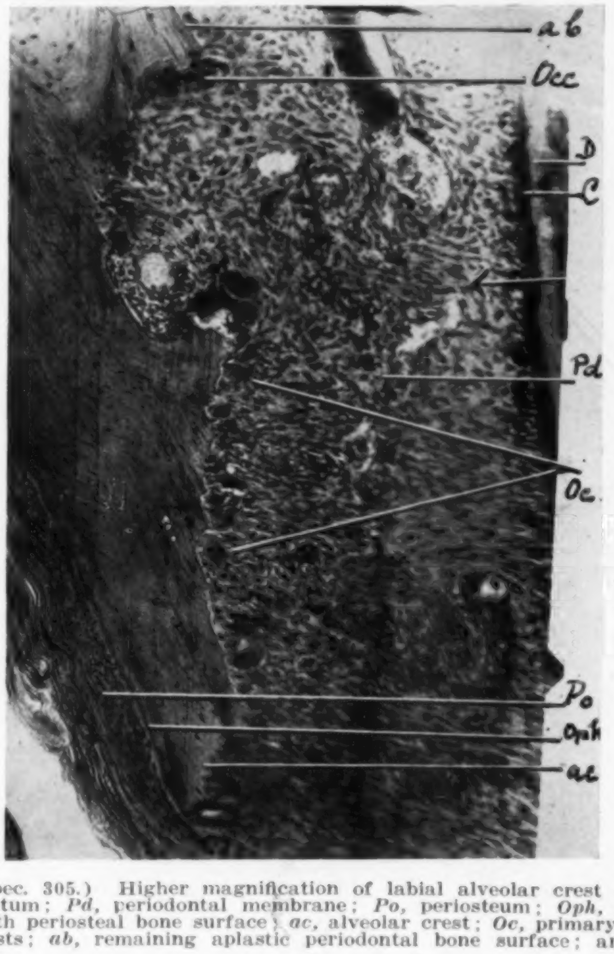

Fig. 32.—(Spec. 305.) Higher magnification of labial alveolar crest (*a*, Fig. 31). *D*, Dentine; *C*, cementum; *Pd*, periodontal membrane; *Po*, periosteum; *Oph*, scarce osteophyte formation on smooth periosteal bone surface; *ac*, alveolar crest; *Oc*, primary osteoclasts; *Occ*, secondary osteoclasts; *ab*, remaining aplastic periodontal bone surface; arrow, direction of primary movement.

By the various factors already mentioned that are responsible for the decrease of force in the progress of movement, the periodontal membrane (*Pd*, Fig. 32) at the crest has recovered entirely. It is wider (0.4 mm.) than on the lingual s. pr. s. (0.2 mm.). In several places above the crest, it is compressed and degenerated, measuring only 0.03 mm. The osteoid bone, formed on the lingual as well as on the pr. tr. s. at the apex (*oB*, Fig. 33, a higher magnification

of *b*, Fig. 31) during the primary movement, has not yet been attacked by osteoclasts after two days of increased pressure, caused by the relapse. The width of the periodontal membrane at the apex is 0.03 mm. on the labial s. pr. s., as compared to 0.4 mm. on the lingual s. tr. s., twelve times as much. Here on the s. tr. s., as well as on s. tr. s. on the crest, we find many osteoclasts still present two days after force discontinuation (*Oc*, Fig. 32). After the same time interval, we found them also in Specimen 254 (*Oc*, Fig. 20).

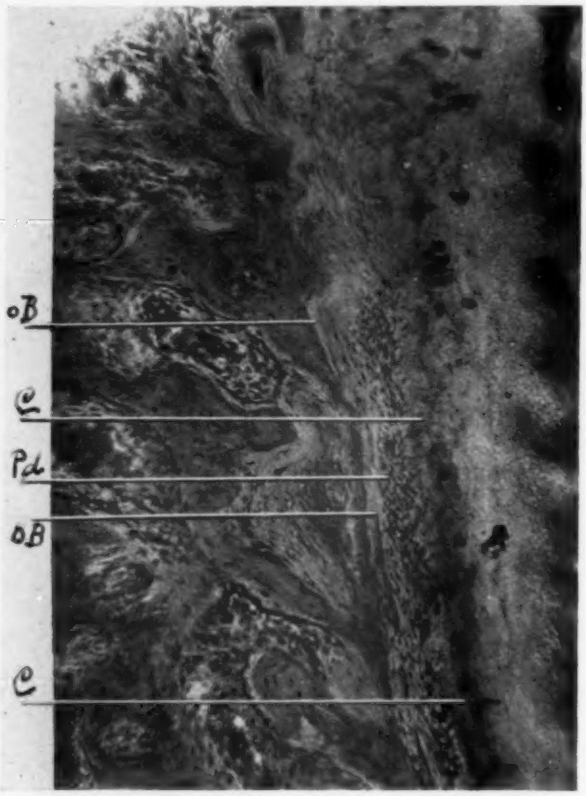

Fig. 33.—(Spec. 305.) Higher magnification of *b* in Fig. 31. *C*, Cementum; *Pd*, periodontal membrane; *oB*, osteoid bone.

The osteophytic bone formation is present but quite poor (*Oph*, Fig. 32). The osteocytes in the primary pressure areas at the crest and apex are normal in the bone that had not been eliminated during the twenty-two days of the primary movement. The pulp in its apical third shows identical changes as described in Specimen 304 (Fig. 29). These changes were also brought about by the irritation caused by the great deviation of the apex, estimated by the difference in the width of the periodontal membrane (twelve times). (See Fig. 31.) The odontoblasts are normal; the cementum is intact.

Fig. 34 (Spec. 303) shows in higher magnification the labial crest of an upper left central incisor moved in this direction with coil springs of the same dimensions and the same compression (240 grams) for twenty-one days and permitted to relapse for three days. The bone (*B*, Fig. 34) became reduced in some places to 0.05 mm. in thickness. The inner portion of the bony plate was eliminated. On the periodontal side of the remaining bone spicule, primary

osteoclasts (*Oc*, Fig. 34) are found three days after force elimination. We found the same persistence in Specimen 253 (*Oc*, Fig. 21) after the same elapse of time. The only sign of a former undermining resorption are excavations of quite considerable depth on the periosteal side, not shown in this illustration. They are located just a little higher up from the alveolar crest (*ac*, Fig. 34). From these excavations the secondary osteoclasts have disappeared and their surface is smoothed out. The osteocytes are normal in the bone that remained.

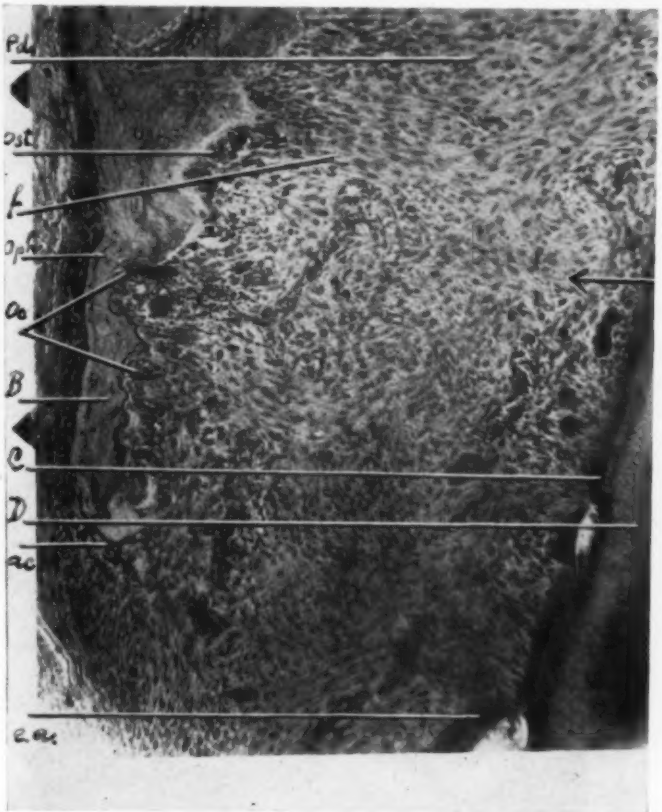

Fig. 34.—(Spec. 303.) Upper incisor; higher magnification of labial alveolar crest. *ea,* Epithelial attachment; *C,* cementum; *D,* dentine; *B,* bone; *ac,* alveolar crest; *Pd,* periodontal membrane; *Oph,* osteophyte; *Ost,* osteoid bone; *Oc,* primary osteoclasts; *f,* strong bundle of principle fibers; arrow, direction of primary movement.

The osteophytic bone formation in this specimen also is quite poor (*Oph,* Fig. 34). After three days of relapse the effect of the traction is very well developed (osteoid seams, *Ost,* Fig. 34), especially where a strong connection between bone and cementum has been established (*f,* Fig. 34). The periodontal membrane (*Pd,* Fig. 34) has recovered entirely. The relapse must have taken place at a somewhat slow rate, for we do not find any crushed tissue on the lingual s. pr. s. Also the difference in the width of the periodontal membrane labially and lingually is not so pronounced as in the preceding specimens. On the lingual s. pr. s., osteoclasts have not yet appeared on the osteoid laid down during the primary movement, though the inceased pressure has lasted for three days. In Specimen 253, they were present after the same time. No pathologic changes are found in the pulp. The cementum is normal.

In Fig. 35 (Spec. 302), we see, in higher magnification, the labial crest of an upper lateral incisor that was moved in this direction with coil springs for twenty days and was permitted to relapse for four days. The periodontal membrane shows normal conditions (*Pd*, Fig. 35). On the scalloped bone surface above the crest (*ac*, Fig. 35), we find primary osteoclasts (*Oc*, Fig. 35). We are still at the beginning of the series, in the region of the sloping root surface where the pressure is not transmitted in full force (Fig. 37) and somewhat above the crest. It is the crest proper which was, and always is, first attacked by osteoclasts. These are the reasons why, from the start, only physiologic osteoclastic activity was at work here on the surface alone. These primary osteoclasts are still present (*Oc*, Fig. 35) four days after force discontinuation. The crest

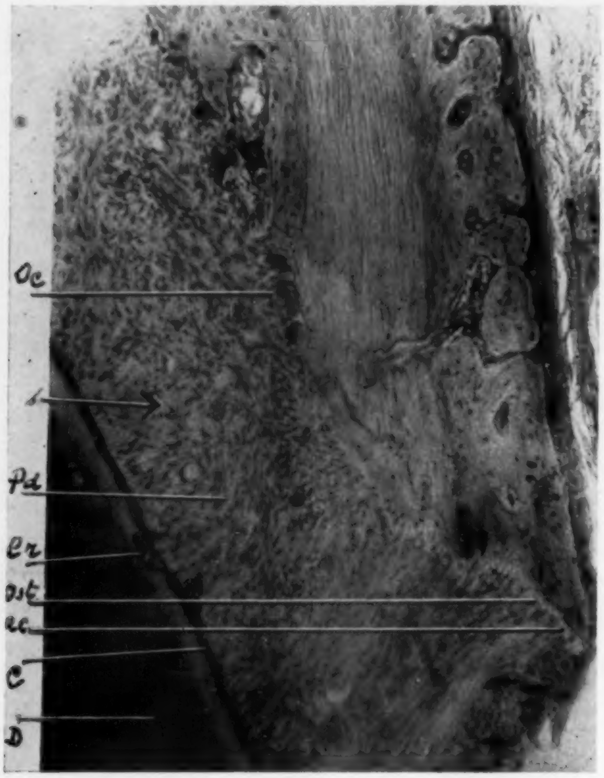

Fig. 35.—(Spec. 302.) Upper incisor; higher magnification of the labial alveolar crest. *D*, dentine; *C*, cementum; *Pd*, periodontal membrane; *ac*, alveolar crest; *Cr*, cementum resorption; *Oc*, primary osteoclasts; *ost*, osteoid bone; arrow, direction of primary movement.

may have been, and probably was, resorbed by undermining resorption, the vestige of which is still discernible at the crest proper (*ac*, Fig. 35), as proved by its blunted appearance. Its surface is now smoothed out and covered with osteoid (*ost*, Fig. 35). No vestige of a periosteal osteoclastic activity can be found. The bone lacunae contain, for the greater part, normal osteocytes; now and then a few empty lacunae are found. The cementum surface is aplastic (*C*, Fig. 35).

In a slide near the center of the tooth, where the root bulges out the most against the bone and against which the full impact is directly transmitted, we find another picture (Fig. 36). Except for the crest itself, the original bone

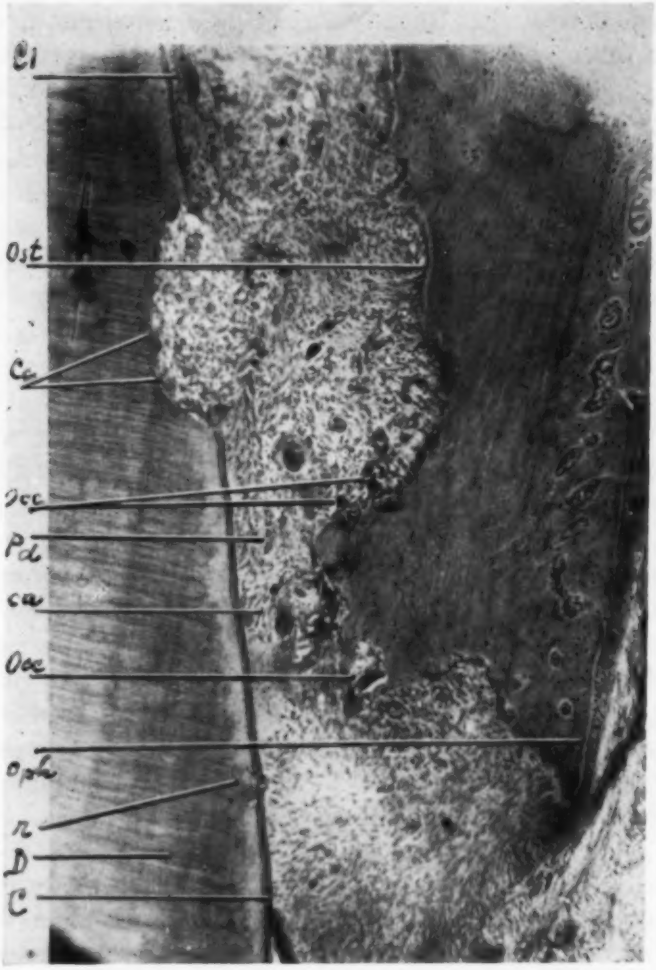

Fig. 36.—(Spec. 302.) Neighboring slide to Fig. 35, more towards the center of the tooth. *C*, Aplastic cementum; C_1, cementum with cementoid seam; *D*, dentine; *Pd*, periodontal membrane; *ca*, crushed periodontal tissue with hyaline degeneration and débris; *Occ*, secondary osteoclasts; *Oph*, osteophytic apposition; *r*, small cementum resorption; *Cc*, greater cementum resorption with cementoclasts still present; *ost*, osteoid.

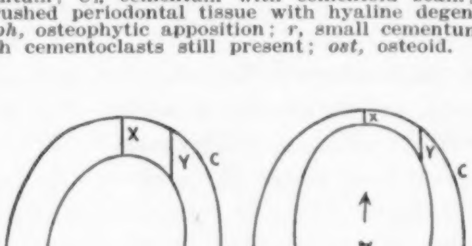

Fig. 37.—Diagram to show how the pressure becomes decreased in the region of the curving root surface; *A*, At rest; *B*, during movement; *r*, root; *c*, alveolar bone; *x*, width of the periodontal membrane in the region of the greatest bulge of the root; *y*, width of the periodontal membrane in the sloping region; at *x* the force is transmitted directly, at *y* in an acute angle; arrow, direction of movement.

and the crushed tissues have not yet been eliminated; therefore, the pressure could not become decreased sufficiently for the periodontal membrane to recover. So, unlike the other specimens, the pressure was still too great to incite the formation of primary osteoclasts. Instead we find the hyalinized tissue and its debris (*ca*, Fig. 36). The osteocytes are mostly normal; the osteophytic bone formation (*Oph*, Fig. 36) is quite poor; no sign of any periosteal osteoclastic activity was found. The cementum within the compression area is aplastic and again displays its signs of vitality (cementoblasts, cementoid seam) above the compression area (C_1 Fig. 36). Within this area, we see a cementum resorption with the cementoclasts (*Cc*, Fig. 36) still present four days after force discontinuation. A proof that the lowering of the crest has really taken place is found in the presence of another small cementum resorption (*r*, Fig. 36) (greater in neighboring slides) in a region opposite which bone is no longer present. The larger resorption, though not deep, is already quite extended buccolingually and can be followed continuously in forty-five slides when the series is interrupted.

Corresponding to these cementum resorptions at the crest, we find another in the pressure area at the apex, where cementoclasts also persisted four days after pressure release. Above the crest we find the effect of the relapse movement of four days, the formation of an osteoid seam (*ost*, Fig. 36). Near the crest itself, osteoid could not form since the osteoclasts are still present.

As in Fig. 23, we find here also after four days of increased relapse pressure that on the lingual s. pr. s., osteoclasts are at work on the osteoid bone surface which developed during the primary movement. The difference in the width of the periodontal membrane in the crest and apex region is considerable here too. At the crest, measurements are 0.2 mm. as against 0.4 mm.; at the apex, 0.1 mm. as against 0.4 mm. on the s. tr. s., four times as much. This indicates a great excursion of the apex. And consequently we find changes in the apical third of the pulp similar to those found in Specimens 304 and 305 (Fig. 29) but not so pronounced.

DISCUSSION

In the beginning, we mentioned that we tried to measure the forces used. In treatment of patients such measuring is useless because the reactions to the same force are different in different individuals.[29] The only criteria that we have as to the appropriateness of the force are the firmness and lack of soreness of the teeth. In animals, the only test is the degree of looseness. Therefore, in order to judge within general limits the relationship between the amount of force and the changes brought about by this force, we had to measure it. For this purpose, Dr. George M. Hollenback, Los Angeles, devised and constructed a small ingenious instrument for which I give him credit and my best thanks.

The movement of teeth, some with coil springs and others with ligatures, was performed to test a previous statement[29] that continuous forces (coil spring) give results different from, and more injurious than, intermittent forces. But this attempt failed because the ligatures were renewed too often (every other day) making their force too strong also; therefore, the results obtained were

quite similar. But where the ligatures became loose or where the force became reduced (e.g., because of the curvature of the root towards the proximal sides), the difference shows up (Figs. 16 and 21). After the use of strong forces we always see the familiar picture of a crushed periodontal membrane, the aplastic bone surface and undermining resorption from either the periodontal or periosteal surface of the bone and the marrow spaces. Quite often a disintegration or disappearance of the osteocytes occurs (Figs. 13, 24, and 28), occasionally of the cementum corpuscles also (Fig. 14). In contrast, where light forces are used the osteoclastic activity takes place only on the periodontal surface, leaving there the signs of this activity (Howship lacunae) (Figs. 16, 20, and 21). But in our present material, even in the specimens under the application of supposedly light forces, undermining resorption starts on the periosteal side (Figs. 11, 18, and 21), and the osteocytes also are attacked (Figs. 13 and 24).

Periodontal Membrane.—In the eight specimens of relapse, we find, in the primary pressure areas, different degrees of recovery of the periodontal tissues, with more or less rearrangement of the suspensory fibers, depending on the degree of alteration during the original movement. The alteration in turn depends on the duration and amount of the force applied. In the case of six days of active movement and relapse of one to four days (Figs. 10 to 25), a recognizable recovery of the crushed tissues did not start until four days after pressure release. In the specimens of twenty to twenty-three days' duration of active movement and one to four days of permitted relapse, full recovery of the periodontal tissues was found. The exact time for the beginning of this recovery of course cannot be given, for it had already started while the teeth were moving. The recovery may depend on various factors: the gradual loss in strength of the spring as it extended itself back toward its original length; the fatigue of the elasticity of the metal; the increase of the periodontal width by resorption of the socket wall and the entire elimination of pressure after force discontinuation.

These findings are nearly in accordance with Gottlieb and Orban,[6] who state that, especially in young animals, undermining resorption is completed and normal conditions restored after ten days in dogs, and from ten to twenty-three days in monkeys. This nearly corresponds to our findings, for in Fig. 36, for example, after twenty days the undermining resorption is not yet completed; but three days may make a great difference. In our specimens it was found that the recovery proceeds at a quicker rate in the apex region than at the crest, apparently on account of better nourishing conditions at the apex.

Marshall[20] stated that it should take five times as long for some tissue changes in man as for similar changes in monkeys. If we assume that Marshall was right, the time necessary for periodontal tissue recovery in man would amount to twenty (4 times 5) days at the apex and somewhat longer at the crest.

As far as the completion of undermining resorption is concerned, it would need (if we take Fig. 36 as a Basis) more than 20 times 5, or one hundred days in man, for after twenty days the undermining resorption is far from being completed. In regard to bone, it has been shown[28] that even under the most favorable conditions, six months were not sufficient for rebuilding and transformation to normal in monkeys. Under Marshall's rule, thirty months

(6 times 5) would therefore be insufficient in man for the bone to return to normal. Soreness and looseness of teeth, clinically indicating nonphysiologic movement, may easily be laid to these two factors: the alteration and destruction, partial or entire, of periodontal tissues, and the great increase in periodontal width on the traction side compared with that on the pressure side (6 and 7 times in Specimens 313 and 310, or 12 times in Specimen 305). This great increase in width on the traction side in the use of strong forces is caused not only by the mechanical increase of the distance between tooth and socket wall, but also by the superficial lacunar osteoclastic activity starting here under the prevailing conditions (Figs. 3, 6, and 8).

Osteocytes.—A compression of the periodontal membrane beyond a certain limit affects the condition of the osteocytes whose existence and survival depend, wholly or in part, on this source for nourishment. The change develops gradually and depends upon the amount of force applied and its duration. It was found far progressed after four days of destructive compression of the periodontal membrane in the use of the coil spring, and at its peak after six days (Figs. 13 and 24, ligature) or more (Fig. 28, coil spring). The force in the use of ligatures was too strong, as stated above. Of the four specimens of twenty-one to twenty-three days' movement, the total disappearance of the osteocytes in the periodontal half of the labial plate was observed in one specimen (Fig. 28). In the other three (Figs. 32, 34, and 35), the part of the bone which may also have been affected has already been removed by undermining resorption. The remaining bone shows normal structure.

The characteristics of these changes are as follows: First the osteocytes start to shrink, become pyknotic, losing thereby their staining properties; they continue to disintegrate more and more till at last only remnants persist or they disappear entirely and the lacunae are found empty. Such a bone has lost part of its vitality dependent on the degree of disintegration of the cells and after their disappearance has to be considered "dead" bone. The osteocytes develop from the osteoblasts during the formative period of bone by their inclusion into the matrix, and once gone they cannot be replaced any more. Such a bone, deprived of its soul, has to be eliminated sooner or later and the amount of loss of bone depends upon the extent of the suffocated periodontal membrane. That this is true is clearly proved by the fact that the osteocytes disintegrate and disappear first near the periodontal bone surface, while deeper, near a marrow space (Figs. 14 and 24), or near the periosteum (Fig. 28), which continue to provide good nourishment, they show no signs of disintegration. The same is the case with the cementum corpuscles (Fig 14). On the traction side they are always found normal.

The osteocytes have disappeared entirely in the periodontal third of the bone in two cases (Figs. 13 and 24) and in the periodontal two-thirds in one case (Fig. 28), that is, in three out of twelve cases (25 per cent). In three cases the cells in the original bone still present were found normal (25 per cent). In the remaining cases (50 per cent) the disintegration was not so severe and only pyknosis and occasional empty lacunae were found. The disintegration of osteocytes is not the same in all specimens treated under quite similar conditions. Sometimes it is found both at the crest and in the apical region, sometimes in

one or the other only. It can only be assumed that it is governed by peculiar local conditions. In places where the osteocytes have disappeared and the osteoclasts have started undermining resorption, it may be presumed that they continue until the "dead" bone (or cementum) is eliminated. This of course could not be proved in these experiments, which ended too soon for that. Orban[31] made the statement "that resorption of bone . . . *can* [italics by the author] continue for months. . . ." Based on the evidence presented here, this "can continue" should be changed to "must continue," for Nature will not tolerate any foreign body, into which category this "dead" bone must be placed. Where the osteocytes remain living, the osteoclasts disappear relatively soon, since no need exists for the removal of "living" bone. But very often, even under these circumstances, the osteoclasts persist for a longer time and "overshoot" the mark (Gottlieb, Orban). Perhaps in these cases the osteocytes really have died too, though this has not yet been proved. Though it may be assumed that the degeneration and disappearance of the osteocytes are not prerequisites to undermining resorption, such an occurrence must be considered an extremely advanced damage to the bone in places of severe destruction of the periodontal membrane. Undoubtedly Nature has to dispose of such a bone.

If we take into consideration the various findings and their explanation by the different investigators in the field of general bone pathology, we will find that all agree as to the causative factors of bone necrosis. Bone is a fibrous tissue which differs from ordinary connective tissue only in that calcium salts are deposited in the ground matrix. A crushed periodontal membrane brings about an inflammation which is bound to spread into the bone. Inflammation mobilizes osteoclasts, which resorb and rarefy the bone (inflammatory resorption). All authors agree in principle that for the death of bone there is one common reason: the cutting off of the blood supply. This may be caused by serous exudation, the proliferation of the usual cells of acute inflammation (polymorphonuclear cells) and thrombosis of the vessels.

"When an area is involved quite suddenly and is of more than microscopic extent, gross death of bone results . . . which corresponds to the amount depleted of its blood supply . . . when such factors are at work all or practically all of a given bone may be devitalized" (Padgett,[34] p. 297, and similar in Boyd,[4] p. 975).

Necrosis of the bone may also be caused by trauma (orthodontic trauma included), infection, or by the action of chemicals (arsenic, phosphorus, lead).

"When the nutrition of the bone is interfered with by disease or injury and the circulation is not re-established within a reasonable time, death of the tissue results" (Mead,[21] p. 388).

The disappearance of the osteocytes, even "their poor staining . . . indicate bone necrosis. Interference with circulation arrests cellular activity . . . and results in necrosis of the bone" (Thoma,[50] pp. 837 and 833).

In orthodontics, when we use strong forces (trauma, injury), we also find the precursors of a developing or developed bone necrosis, singly or combined. We find the clinical signs for such happenings, the soreness and looseness of the teeth, and the morphologic histologic signs: inflammation, serous exudation, accumulation of the inflammatory cells, acute osteoclastic activity, vessel thrombosis, hemorrhages. In addition, we find one of the first and most certain

morphologic signs of bone necrosis, the heretofore unobserved faint staining, crumbling, and disappearance of the osteocytes.

In a far-advanced stage of bone necrosis after the formation of a sequestrum, Thoma[50] (p. 832) observed "that the bone cells had degenerated."

This death of the bone cells is also mentioned by Mead[22] (p. 845): "In caries of the bone (rarefying osteitis or osteoporosis) the cells die successively or piecemeal, being a molecular destruction."

Leriche and Policard[18a] express their view on this point in their book (p. 49) admitting thereby, in accord with the other authors, that dead cells mean dead bone: "Physiologically, dead bone, with dead cells, seems to have the same mechanical value as when it is living. But as long as it is *tolerated* [italics by the author] and as long as it is maintained in the organism, it seems to play the same mechanical part."

So far, the formation by granulation tissue of a demarcation line between the live bone and dead bone and their separation by the formation of a sequestrum has not yet been observed in our field. If a sequestrum is small (and in our field it never can reach greater dimensions), it usually becomes resorbed in time. After all, we have to deal nearly always with a traumatic aseptic inflammation.

The disappearance of the osteocytes has not been reported previously but from a notice just received from Orban, Sicher, and Weinmann,* who also examined the specimens at hand, we learn that such empty bone lacunae in areas of high compression of the periodontal membrane can be seen in Fig. 48 (dog) and Fig. 192 (human) in Gottlieb and Orban's book,[6] but had been overlooked. The human specimen is also reproduced by Orban[33] (Fig. 10). In re-examining their material, they could verify these findings here reported in dogs as well as in human beings. This occurrence may be considered now a well-established fact in dog and monkey as well as in human beings.† This disappearance of the osteocytes, this dying of the bone, may also have far-reaching consequences. Though the periodontal membrane recovers relatively quickly, it is not certain that, after regaining their vitality, its cells will resume their work to lay down new osteoid, on "dead" bone. If this does not happen, an imbedding and reattachment of the suspensory fibers cannot take place until this "dead" bone is eliminated. During this time (which may last for months) no transmission of the functional stimuli can take place, which is a well-known reason for the disappearance of bone, even if only locally, impairing correspondingly the resistance of the tooth even to stresses of normal function. The damage done during this period is apt to lower the resistance forever, the consequences appearing much later (paradentosis).

Osteoclasts.—In the specimens of various areas secured in the course of these experiments, three kinds of osteoclasts are found, each having a different significance though the same purpose. Where no crushing of the periodontal membrane occurs and the vitality of the tissues is not interfered with, the physiologic reaction against increased pressure takes place. It was shown by Schwarz[44] that even with a force of 3 to 5 grams, teeth can be moved a distance

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†By re-examining his own human material, the absence of osteocytes could be verified by the author, too; it was also overlooked and not reported in the original publication.[39]

of 1 mm. in one month.* Osteoclasts are brought into being and act only on the surface producing a uniform lacunar resorption, the ideal reaction. The osteocytes maintain their morphologic characteristics and do not disappear. From the standpoint of the general pathologist, this ideal reaction is confirmed by Leriche and Policard[18a] (p. 82): "From a practical point of view, only well-vascularized bone, that is, living bone, is capable of being quickly resorbed." These osteoclasts, which work relatively quickly and for some time continuously, we will call *primary osteoclasts* (*oc*, Figs. 16, 20, 21, 23, 32, 34, 35, and *ocl*, Fig. 27).

One may consider it a rule wherever primary osteoclasts are at work, that the marrow spaces in the neighborhood never contain osteoclasts in a number worth mentioning; there is either the normal lining of the endosteum with more or less numerous osteoblasts, or compensatory osteophytic bone formation takes place (Fig. 20, *oph*). But where the tissues became crushed, no osteoclasts, therefore, could be formed in the immediate neighborhood (especially in widespread crushing). Then very active osteoclastic activity starts from the marrow spaces (*Occ*, Fig. 13).

In his experiments, Schwarz[45] (p. 350) found "that the most favorable treatment is that which works with forces not greater than the pressure of the blood capillaries. This pressure in man, as well as in most Mammalia, is 15 to 20 mm. Hg; it is about 20 to 26 grams to one square centimeter of surface . . . such a pressure is so intensive that a continuous more or less lively resorption takes place in the alveolar bone at the region of pressure."

The life span of osteoclasts, the time during which they remain active even though the increased pressure which caused their presence has subsided, can be judged only about primary osteoclasts. With the specimens at hand, no definitive answer to this question can be given, because the duration of the experiments was too short. So far, their presence could be ascertained beyond a doubt even four days after force elimination (*oc*, Figs. 34 and 35).

The persistence, of osteoclasts for as many as thirty days was reported by Skillen and Kriwanek,[37] though it must be admitted that their persistence was traced to an inflammation of the gum brought about by bands and ligatures. Each inflammation of the gum, acute or chronic, is accompanied by some bone involvement at the crest (horizontal atrophy).

The same phenomenon of persistence of osteoclasts was also observed by Gottlieb and Orban; they coined for it the expression "overshooting the mark." These authors do not state a definite time for this persistence. They[6] speak only in general terms (p. 196: "The resorption continues even if the width of the periodontal membrane has increased to quite a considerable degree."

In human material, the author reported the presence of primary osteoclasts seven days after the last force application by means of ligature (Fig. 9).[29]

Ziebe[51] made the observation in dog experiments that "the movement of the teeth, as well as the transformation of bone, continues for a long time even if the direct influence of the appliance has subsided." Mershon[23, 24] came to the same conclusion of continued movement by observation in hundreds of cases in

*The forces used in the specimens at hand amounted to 240 and 360 grams, respectively.

the practice. Brodie,[5] also, had the same experience. In concluding his views on this question, he says (p. 215), "It is a common clinical observation that teeth will continue to move long after such movement has reached the apparent limit or extent of the adjustment. This is tissue reaction, and to date we have no evidence to guide us in our judgment as to how frequently we should renew forces."

Maybe the views and proofs given in this paper will serve as a satisfactory answer. At least they prove we, too, are "overshooting the mark"; we are doing much more than is necessary to reach our purpose; on the contrary, we delay it; we are practicing polypragmasy (overdosage). Also, Hemley[13a] will find an answer to his question (p. 330), "How long should pressure be applied and how long should be the intervals of remission?" Where the periodontium has changed in a pathologic way and the osteocytes have become disintegrated or have disappeared, this bone, doomed to elimination, becomes attacked from all sides; from the neighboring uninjured periodontal surface, from the marrow spaces or the periosteal side or from all sides simultaneously. The start of a quick resorptive activity near a crushed or disintegrated area is attributed by some authors to the irritation by the toxins from such tissues. The prolonged presence of the osteoclasts in such an area is necessary and understandable. These we will call "secondary osteoclasts" (*occ,* Figs. 22, 23, 32, and 36, and *oc,* Fig. 27). In areas where the nourishment of the bone from the periodontal membrane has been impaired to a greater or less degree, these secondary osteoclasts develop on the periosteal side to help remove the affected bone as quickly as possible (*Occ,* Figs. 1, 2, 5, 9, 18, 21, and 27, and in Specimen 303). It is quite remarkable that secondary osteoclasts on the periosteal side are already present after one day, maybe even sooner (Fig. 1). Gottlieb and Orban[6] reported in their dog experiments that osteoclasts appeared after one day and even as early as twelve hours after pressure application on the periodontal bone surface. By the combined activity of the secondary osteoclasts, the thickness of the labial plate near the crest became reduced to 0.05 mm. in one instance after four days of movement (Fig. 9), and after twenty-one days in another (Fig. 34). In the first case, the force of the spring was 360 grams, in the latter, 240 grams. The normal width of the labial plate in monkey incisors according to histologic measurements of six normal specimens amounts to an average of 0.27 mm. The alveolar crest has disappeared entirely in Specimens 252, 305, and 302 (Figs. 22, 32, and 36).

Based on their abundant material, Gottlieb and Orban[6] make the statement (p. 145) that "this (viz. disappearance of bone at the alveolar crest) is an apparently lasting damage and hardly any repair follows."

In dog teeth on the periosteal side near the crest, osteoclasts were seen by Gottlieb and Orban (Fig. 24)[6] after thirty-six hours of pressure. Not enough emphasis was laid on this occurrence which, in our opinion, should be considered chiefly responsible for the disappearance of the crest.

Such a periosteal osteoclastic activity was not reported in the experiments of other authors either on animals or on human material. (On dogs: Schwarz,[43, 44] Skillen and Reitan,[38] Oppenheim;[27] on dog and human: Stute-

ville,[41] Orban,[31] Skillen,[39] Gottlieb;[11] on monkeys: Johnson, Appleton, and Rittershofer,[15] Oppenheim;[25] on human: Herzberg,[14] Oppenheim.[29*])

To sum up the whole situation: Secondary (periosteal) osteoclastic activity is found beyond any doubt in nine out of twelve cases (75 per cent); in one case (Fig. 22) nothing certain can be said for the crest has already disappeared. Only in two specimens (Figs. 32 and 36) is there no indication of such a resorption. Why these resorptions, irrespective of the amount and the duration of the force, are sometimes more shallow and extended, in other cases again more deep with limited extension, cannot be explained; maybe local reasons have to be blamed. However, the absence of this peculiar condition in man should not be looked upon as a more favorable condition; maybe it was overlooked on account of the scarcity of human material. Now that attention has been called to such happenings, it is probable that similar reactions to the same cause may be found in human, too. The periosteal resorption as well as the disappearance of the osteocytes may serve as an additional warning against a too great or a too long protracted compression of the periodontal membrane.

After the establishment of a crushed periodontal area and the start of an undermining resorption, what does Nature do, and what the orthodontist? Once a tooth becomes blocked, its movement comes to a standstill. At the end of several weeks, Nature through osteoclastic activity has removed the "dead" bone and the crushed tissues as described; the tooth again makes a movement, though not always just in the desired direction. Then, if the same force is still acting or has been renewed, the same play starts again; the tooth moves by fits and starts.

This is why, even in the use of strong forces, results are obtained, although much valuable bone is lost, sometimes irreparably.

What does the orthodontist do? If he cannot observe any progress for several weeks, he renews or increases the force, blaming its seeming weakness for the standstill. This results only in a broadening of the area of destruction. The consequences of this procedure manifest themselves only years after treatment is finished (paradentosis). We can merely repeat once more: In encountering greater resistance, the right procedure should be to double the time, not the force.

Tertiary Osteoclasts on the Traction Side.—In the use of light forces when no fibers are torn, we find an even smooth layer of osteoid (Fig. 19). When stronger forces are used and only a few fibers are torn, the bone follows in elongated spicules (Fig. 12) in places where the connection between the cementum and the bone remained intact. In the use of strong forces, most of the fibers are broken and only now and then a trace of osteoid formation is found. Many of the vessels are ruptured and hemorrhages are found everywhere (Figs. 3, 6 and 7, and Specimen 304), more or less numerous and pronounced in different specimens. By the severing of the vessels, the nourishment of the bone is interfered with; its vitality is correspondingly lowered. By the decomposition of the red blood corpuscles, toxins originate. Both the lowering of the vitality and the toxins are responsible for the mobilization of the osteo-

*Quite a number of publications dealing with the experimental elucidation of different orthodontic problems are not enumerated because they are concerned with other problems.

clasts which resorb the bone not by undermining resorption but only from the surface or the marrow spaces; occasionally from both sides. This condition was found more or less pronounced in all the four specimens of the first series (coil-spring action of 360 grams).

The resorption of the bone sets in from causes explained above though morphologic changes in the osteocytes cannot yet be ascertained. The nourishment of the bone is only reduced, not cut off entirely as on the pressure side. This may be responsible for the slower disintegration of the osteocytes; for not all vessels are torn and the remaining provide the pabulum—though at a decreased rate, thus maintaining the vitality of the bone at a certain level.

The osteoclasts at the traction side should be ranked with secondary osteoclasts though they act on the bone surface (Figs. 3, 6, and 8) facing the periodontium, like the primary osteoclasts under the application of light forces (Fig. 16). For these osteoclasts the author proposes the name "osteoclasts of the third order" or "tertiary osteoclasts." Such osteoclasts cannot be found in the use of strong forces on the pressure side because, by the crushing of the tissues, no cells are present from which they could originate—therefore we find resorption from all sides (undermining) except from the surface.

Under the application of strong forces, we find osteoclastic activity on both the pressure and traction side; on the pressure side, the destructive undermining resorption; on the lingual side, the superficial lacunar resorption. The great looseness of teeth must be the result of their combined activity plus, of course, the mechanical widening of the periodontal space by the traction. This looseness is not reduced as under application of light forces—by the deposition of osteoid bone, that normally develops simultaneously with the starting movement —Nature's way to maintain the normal width of the periodontal membrane. But under the application of strong forces this osteoid cannot form for two main reasons: (1) No stimulus can be transmitted to the bone, the connection between tooth and bone being severed. (2) Where osteoclasts are at work—and the reasons for their presence were given—no osteoblasts can work simultaneously to produce the osteoid.

To sum up: The characteristic tissue changes under the use of strong forces on the pressure side are: hyalinization, destruction and disappearance of the tissues of the periodontal membrane, with accompanying resorption of the bone from all sides except the side of the primary force impact (undermining resorption). On the traction side the tissues preserve their vitality and staining properties (at least for a while); many fibers are torn, many vessels ruptured, and hemorrhages formed. In rare instances only, scanty osteoid bone formation is found; on the contrary, the bone becomes resorbed by superficial lacunar resorption still more widening the periodontal membrane.

Intermittent Light Forces and Osteoid.—In repeating a review of the Andresen Method[1] by the late Axel Lundström (INTERNATIONAL JOURNAL OF ORTHODONTIA, November, 1936), the British Dental Journal quotes "on account of its importance" from this review, as found in the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY, September, 1943, page 564: "A method using appliances to be worn at night has such decided advantages that we should consider the possibility of using it for every patient."

The main objection to such a procedure is the supposed lack of cooperation and the possible loss or distortion of the appliances. But these objections, especially the lack of cooperation, are not justified. Even with fixed appliances when changes in mesiodistal relationship are needed, we depend on full cooperation; without it we will get nowhere. The same applies for the use of extraoral appliances so much in vogue now. It takes the authority, sincerity, good will, and capacity of the orthodontist to impress the patient with the indispensability of his cooperation and the many advantages of such a procedure. If full cooperation cannot be assured, the treatment should be discontinued just as with fixed appliances.

The only disadvantage may be the longer "treatment" time; but this is many times compensated for by the ease, comfort, painlessness, by the absolute mouth hygiene and by the better prospects for permanency both of the immediate accomplishments and the results after discarding all appliances.

If we look at the question from another angle and compare the number of hours spent in actual treatment in cases of day and night treatment (twenty-four hours) with that of night treatment alone (ten hours), we will see that as far as tissue strain is concerned, there is an advantage in the latter. If we assume that on an average a treatment with continuously worn appliances is performed in one and a half years, or, as in most cases, in two years, then 13,128 or 17,520 hours, respectively, were spent. Such a treatment is usually performed without rest periods.

If, on the other hand, we assume that a case with night treatment alone needs for its completion, roughly, four years, then 14,600 hours were spent in actual treatment; and it is not too much to assume that during these four years, on an average, rest periods totaling four months were interspersed; that is, ten hours for 120 days, or 1,200 hours, were eliminated; the actual treatment time therefore amounts to 13,400 hours; it is only 272 hours (11 days) more in comparison to a continuous treatment of one and a half years and it is less by 4,120 hours (172 days) when the continuous treatment lasts two years. This means considerably less strain on the tissues and therefore better chances for repair and restoration. These facts were corroborated in practice and are reported in the second part of this paper.

It is the author's belief, based on many years of observation, that Schwarz[46] was right in stating that "through orthodontic intervention paradentosis might be started, since orthodontic measure means overstrain and this can cause paradentosis;" but it is also our conviction that these bad aftereffects can be reduced to quite a considerable degree when the orthodontic treatment is performed along lines that insure a more physiologic reaction to our measures.

Lehner and Plenk[18] had the honor of writing the chapter "Teeth" in the leading *Moellendorf's Textbook of the Microscopic Anatomy of the Human,* and though not orthodontists, summed up their conclusions from microscopic findings (p. 661): "The secret of the unexpected results of dental orthopedia lies in the use of natural biologic methods, in the use of which the application of force is avoided and the natural processes of apposition and resorption in the paradentium are made serviceable for the regulation of teeth."

It has been shown by several writers[6] and by the author himself[30] that osteoclasts develop quite readily on the original pressure side even as soon as twelve hours after stimulation. This paper offers further corroboration. However, we found that such osteoclasts do not develop during the relapse on the secondary pressure side (traction side of original movement) after one and two days of increased pressure against the osteoid formed there during the primary movement. It took three days (Specimen 253) and four days (Specimen 302 and Specimen 252, Fig. 23, *oc*) before the osteoclasts made their appearance. But in Specimen 303, the osteoid did not become attacked after three days of increased pressure (relapse). This late appearance of osteoclasts may be accounted for by two factors: either the relapse and the compression of the tissues has occurred too quickly, thus reducing or arresting entirely the potentiality of the connective tissue cells to transform into osteoclasts, or it might have been impossible for the osteoclasts, already present, to attack at once the young uncalcified tissue. This greater resistance of osteoid bone has always been maintained by Gottlieb,[9] Gottlieb and Orban,[6, 7] (Fig. 143), Orban[31, 32] (Fig. 88), Kronfeld[16, 17] (page 384), Sicher[47] and others. Though it, too, becomes resorbed at last, as was shown previously,[29] the author must admit now that he was wrong in maintaining that there is no difference at all between mature, well-calcified bone and young bone. The time factor counts; a longer time is needed before the osteoid becomes attacked by osteoclasts.

Reasoning from these facts, light *intermittent* forces, as always advocated by the author, now would not be, theoretically, the right procedure because of the osteoid formed during the intermissions. The use of light *continuous* forces would seem to be the right way, thus eliminating such osteoid formation. But this could only be attained if the movement would keep pace with the bone resorption. The width of the periodontal membrane, continually reduced by the appliance force, thus would be maintained continually at its optimum, and the greatest number and the greatest possible activity of the osteoclasts would be assured. However, even with the lightest intermittent forces, we found it impossible to achieve in man such a uniformity of movement and resorption, because, with the appliances at our disposal, we can never approach the slowness of physiologic movement. Even before the end of ten weeks, in man the signs of pathologically quick movement, with all its consequences, developed.[29] If the same coordination between movement and bone resorption could be established in orthodontic treatment as is found during the continuous mesial drift of the teeth by wearing down of the contact points, this would not only guarantee a continuous superficial bone resorption but also the integrity of the cementum. The protagonists of continuous light forces are Mershon,[23, 24] relying on practical experience, Schwarz,[45] Gottlieb and Orban[6] (p. 224), and Orban,[31] who based their arguments on histologic evidence. However, Gottlieb[11] admits now the practicability of intermittent forces. He states (p. 172), "If our treatment causes tooth resportion and we wait till a repair layer of cementum takes place with reconstruction of the periodontal connection with the bone, then we may go ahead with the movement." On another occasion[10] after having discussed why the strong forces of mastication are not injurious to the tissues of the paradentium, Gottlieb concludes: "The lesson learned thereby for orthodontic

procedures is to give frequent opportunities to the tissue for recovery and not to continue with unbiologic forces for months and years."

The justification for intermittent forces is found in the histologic findings here reported, in the clinical observations of the author and others (Brodie, Gottlieb and Orban, Mershon, Ziebe) and further observation by some practitioners in California (to whom credit will be given in the second part of this paper).

We refer to "intermittent forces "in the new sense, not in the old meaning where the osteoid formation took place during the successive adjustments and force renewal. As far as inferences may be permitted from monkey material to human,* the way to perform physiologic tooth movements (as corroborated by practical proof in the second part of this paper) would be as follows: Use a stimulus for a short time (one day or one night only) followed by an intermission of the same duration or even of two or three days; *a new stimulus should not be applied as long as the primary osteoclasts mobilized by the preceding stimulus are at work.*† This would produce an even superficial lacunar resorption, similar to that found on the mesial side of the roots during their continuous physiologic mesial drift. The osteoclasts would work in the direction of the intended movement only. The tooth would move as a one-armed lever, no fulcrum having been established. The axis of movement would lie at or near the apex, which would undergo no deviation or, at least, none that was appreciable. The otherwise unavoidable injuries to the pulp would accordingly be reduced. Such a movement would surely also be a prophylactic against the cementum resorptions. It may be assumed that such a procedure would prove not only relatively the quickest but also the most gentle movement, since, according to histologic evidence, primary osteoclasts develop in greatest number under application of light forces (stimuli) and continue to work for at least four days. (In one human specimen, as already mentioned, active osteoclasts were found even seven days after the last ligature renewal.)

By application of the force in this way where movement would keep pace with resorption, no fulcrum would be created, the tooth would move as a one-armed lever, and no harmful deviation of the apex would occur. Even in the use of light intermittent forces in the old sense, we saw[29] that after a certain time (about fifty days) the establishment of a fulcrum near the crest was inevitable with subsequent remarkable deviation of the apex.

The only question is whether orthodontists would find the outlined procedure practical as well as effective. At any rate, the method is worth trying. The responsibility for evolving mechanical appliances harmonizing with the results of this investigation rests with the ingenuity of the members of our profession. However, appliances meeting some of these requirements have been designed for certain cases by the author and have proved satisfactory. Some of these will be shown in the "practical part." Such appliances were designed for (1) the distal movement of the buccal teeth in the maxilla in Class II cases

*That such inferences on account of the new findings in human material also, are not very restricted was already shown.

†In practice it has been realized for many years that osteoclastic activity must have continued after force discontinuation; otherwise no results could have been obtained in the use of the head cap. But only by these experiments is the proof given that this is really true, and are now explained the astonishing results obtained by the application of intermittent forces, intermittent in the new sense.

with superior protraction; in cases with mesial drift of the buccal teeth into Class II relationship, crowding out canines; in Class I and Class II cases to prevent the creation of a double protrusion (head cap); (2) straightening out the curve of Spee by the use of a bite plate; (3) the correction of Class I cases with inlocked upper incisors simulating a Class III condition; (4) moving any teeth buccally or labially by the force of the expanding gutta-percha interposed between a plate and the teeth. These forces, however, may prove too strong, for, in one case (the only one in the orthodontic literature), the force of the expanding gutta-percha caused a cementum resorption in a premolar[29] (Fig. 89). But such an experiment has to be repeated, for no conclusive inferences are permitted from one incident only. All these methods were reported in the *Angle Orthodontist*.[29]

It is a well-known fact that most of the destructive changes, even those brought about by strong forces, are for the most part repaired to normal. But one part which is never restored to its original height is the buccal or labial alveolar crest. This is the very part of the surrounding and protecting bone with which we should be most concerned. This loss of bony support which cannot be ascertained by x-ray is responsible for gum recession and exposure of the root. The lasting firmness of the teeth is dependent on the height of the alveolar bone. If this height becomes reduced prematurely, even the normal forces of mastication ultimately prove detrimental. At the end of a somewhat prolonged treatment, the dental age of the patient no longer corresponds to his chronological age. We have established a premature senescence of the denture and thus started the development of, or at least the predisposition to, paradentosis.

Osteophytes.—Osteophytes, usually well developed in monkeys after five days[28] and in dogs after eight days,[6] were found quite poor even after twenty-three days in the specimens presented here. Good examples of osteophyte formation are given in Figs. 11 and 20, *Oph.* It happens sometimes that the old bone is replaced entirely by osteophytic formation that now borders the periodontal space; it may attain the same or even a greater thickness than the original bone. It is gradually transformed into lamellated bone. The osteophytes in man usually develop very slowly and apparently need considerable time for their formation. This is another important reason for advocating slow movements. Let Nature have plenty of time; give her a chance to compensate for the thinning of bone caused by the resorption.

Cementum.—In the areas of crushed periodontal membrane, we find the cementum surface, like the bone surface, aplastic. The cementoid seam and the cementoblasts, the signs of high vitality (Gottlieb[8]), have disappeared but make their appearance just above and below these areas (as in Figs. 1 and 36). This lowered vitality may mean a predisposition to resorption and may be considered as its precursor. The cementoid, like the osteoid on bone, seems to be a protection against quick resorption. And even more than that, according to Gottlieb,[8] a cementum with lowered or lost vitality may be considered able to stimulate the creation of osteoclasts. "Each disturbance in the formation of cementoid, that normally takes place all during life, has to be considered as a pathologic condition" (Gottlieb,[6] p. 202). In some specimens we noticed also the disappearance of the cementum corpuscles in the superficial layers near the

periodontium (*eC*, Fig. 14), while in the depth they were found intact, just the opposite to normal. As in bone, the impaired nourishment of the superficial layers has to be blamed. The disappearance of the cells, the transmitters of the nourishing fluids, has to be considered as a loss of vitality, or at least as a lowering of it, depending on the degree of the disintegration and the number of cells involved. The author[30] found that, in man, cementum resorptions developed after application of artificial forces of even short duration (one day) and, therefore, they must be considered as an unavoidable occurrence. These findings lead the author to repeat his statement made previously[29] that human cementum has to be considered a quite vulnerable structure. Since all other authors agree now that cementum resorption is an unavoidable occurrence, it can only be assumed that all forces used, even the supposedly light ones, were too strong or that not sufficient periods for recovery were interspersed. The method as outlined above may prove a prophylactic way. In the twelve monkey specimens at hand, we found cementum resorptions on only three occasions, and even these were shallow and not very extended (Figs. 20, 22 and 36). It took six days (Fig. 22), maybe less, before the cementum resorption developed.

As in bone, the resorbing cells were found still present four days after force discontinuation (*Cc*, Fig. 36). This persistence of cementoclasts was found by Gottlieb and Orban[6] in a monkey molar after fourteen days and even longer in dog teeth. Such findings caused these authors to say that "cementum resorptions as a whole are uncalculable" and Orban[32] made the statement, "Sometimes resorptions are not repaired but may continue. . . ." In the same work[6] (Fig. 194), these authors show a human tooth with continuing cementum-dentine resorption.

It is quite astonishing that these surviving cementoclasts are found mostly at the deepest points where, on account of a greater periodontal width, the pressure has become reduced, while near the border with a narrower periodontal membrane, repair is already setting in. Near the apex where the distance to the pulp is sometimes quite short, this persistence of the resorbing cells may occasionally prove dangerous to the pulp, as shown on previous occasions.[29, 30]

Pulp.—In some specimens, remarkable changes were found in the apical third of the pulp. The pulp tissue is transformed into a structure more like connective tissue. These changes were not found until after twenty to twenty-three days of movement (maybe sooner) and were caused by the great deviation of the apex (Specimens 302, 305, and 304, Fig. 29). This deviation was estimated by comparing the difference in width of the periodontal membrane on both sides of the apex. In some instances it amounted to six or seven times (Specimens 310 and 313), and in one instance (Specimen 305) even to twelve times, after twenty-two days of movement. As to the chances of the pulp returning to normal we are not in a position to state.* The span of the experiments was too short.

In his paper,[15a] Kaletzky states: "The dental pulp may withstand a severe trauma and often will fully recuperate," and, "It is not surprising to learn of

*Occasionally there were found some changes other than those mentioned but they were not reported on account of their possible or probable presence caused by the artificial opening of the pulp just before the animal was sacrificed. Only findings were reported that could be traced beyond any doubt to the application of the artificial force.

the actual recuperative powers of the pulp." But in concluding his article Kaletzky says: "I hope this presentation will not encourage the use of too drastic procedures in the movement of teeth [on account of the reported recuperative powers of the pulp], for, surely, there must be a limit somewhere to the amount of insult any tissue may take."

Relapse and Self-Alignment of Teeth.—It is a common observation that, the quicker the original movement, the quicker and more forcible the relapse. This is easily understood when we consider the various forms of osteoid formation on the primary traction side. We know that osteoclasts develop as the specific reaction of the connective tissue cells against increased pressure. The specific reaction against increased traction is the formation of osteoblasts and osteoid. In the use of strong forces, no osteoid is formed on the traction side; on the contrary, osteoclasts develop instead, resorbing the bone here too (Figs. 3, 6 and 7).

Gottlieb and Orban[6] showed that formation of osteoid bone had started on the traction side after twelve hours, and that this formation after five days had caused a remarkable narrowing of the periodontal space. In the course of a slow movement, the osteoid forms in an even layer all along the bone surface (***Ost,*** Fig. 19), while during quicker movement, only several elongated spicules are formed in places where the principal fibers between bone and cementum have not been torn (Fig. 12). Ziebe[51] has also demonstrated that where light forces were used, the fibers under tension not only become more numerous but also thicker and therefore less liable to rupture. This increase in number and strength of the fibers in order to give a better attachment to the tooth in cases where the occlusal stress becomes greater than normal, as in traumatic occlusion, is several times reported. Such a reinforcement of the fibers assures the formation of an uniformly thick layer of osteoid that will offer resistance greater than just a few elongated spicules could. Since the osteoid has a longer time to form, it is thicker, and so the space available for relapse is diminished. Besides, newly formed osteoid offers a greater resistance to resorption.

With all of these conditions prevailing, we still have a number of relapses; hence, the suspicion (for we do not know positively) may be justified that our presumably light forces are still too great. For, if we have to deal with crowded and rotated front teeth, especially in the maxilla, and simply make room by widening or lengthening the arch, as may be needed, retaining the space gained[28] (1934, p. 763), these teeth arrange themselves into perfect alignment through the natural forces of the tongue and lips and never relapse; these forces may be helped by appropriate exercises (Rogers). (See Figs. 40, 51, 54, and 55.)

Of course there are other reasons for relapse. Relapse after individual tooth movement may be due to local causes while, on the other hand, general and recurring relapses (Class II) "may be due to anomalies in growth, which cannot be counteracted or overcome. . . " (Hellman[13]).

The uncertainty of maintaining the results obtained brought Skogsborg[48] to advocate surgical measures, with the guarantee of a permanency of the results after cicatrization of the soft and hard tissues. But this method quickly proved to be a failure and fell into discredit.

The surest way still seems to be only to "help" Nature, to use appliances only for the most necessary, unavoidable movements, and to leave some work for Nature herself to do. Of course this takes more time, but only seemingly so, because we surely save much more time by eliminating, or at least reducing, the chances for a relapse. But even with our present knowledge of tissue reaction and our practical experience to justify the hope for good and permanent results, there are still certain cases that resist treatment. They yield neither to strong nor to light forces. All our endeavors are in vain. We know nothing about this reluctance to respond normally. Though these cases are quite rare, it is safest never to promise a result at the start of treatment but to postpone a prognosis (as far as the active treatment is concerned) for several months, until there are undeniable signs of tissue response, signs that the teeth really are moving.

The number of such cases among those 500 to 600 treated by the author may amount to approximately ten cases, or 2 per cent (exact numbers cannot be given for the records are unavailable). Lundström[19] reports that in 5 per cent of his distoclusion cases he could achieve no result and in 12 per cent only a partial result.

Why, in some cases, we can obtain no result at all, why among quite similar cases, even in twins, some stay while others continually relapse, why the head cap works sometimes only on one side, all these are topics still open for further investigation.

The reason why in the experimental work, one so seldom gets physiologic tissue reactions, and why, in some rare instances, we still get them, is easily explained by peculiar anatomic conditions. If the pressure is applied in a direction that will meet in straight transmission the greatest bulge of the root, the greatest impact will be in this region. The force becomes gradually reduced in approaching the approximal surface on account of the relative and absolute widening of the periodontal membrane by the curvature of the root surface; the force becomes further reduced by its transmission in an angle (see sketch, Fig. 37). Anatomically conditioned, therefore, the pressure becomes reduced and not too seldom we approach or even obtain in these regions physiologic reactions (Fig. 16). Why, besides this, we get this physiologic reaction in one place and not in the nearest proximity is also anatomically conditioned and well explained by Fig. 23. Though we are not dealing with appliance action, we still have to face a relapse of a tooth moving at a much quicker rate than under normal physiologic conditions.

The normal approximate width of the periodontal membrane is 0.2 mm.; the tissue is soft and easily compressible; as connective tissue it possesses the inherent potentiality to react against increased pressure by the formation of osteoclasts; exclusively on this tissue and on its reaction depends the possibility of tooth movement; in a cadaver we can extract a tooth but we can never move it. If we keep in mind that in our work we have to deal always and constantly with this soft tissue of only 0.2 mm. in thickness, it is easily understandable why a compression of a tiny fraction of 1 mm., more or less, will already yield quite different results. At *Oc*, in Fig. 23, we see the physiologic tissue reaction against the increased pressure of the relapse movement; but in

adjoining spicules at *n*, Fig. 23, the pathologic effect of this same relapse is manifest. The crushing of the periodontal tissue and the start of undermining resorption (*Occ*, Fig. 23) are caused by an infinitesimally small projection of these spicules into the periodontal membrane, thus reducing the space between bone and tooth. The width of the periodontal membrane at *Pd*, Fig. 23, is 0.13 mm., at *n* it measures 0.0183 mm. The difference of 0.1 mm., imperceptible to the naked eye, is responsible for the different reaction, physiologic on one place and pathologic on the other. What a discrepancy between the width of the periodontal membrane and the distances we move teeth! And this unrealized discrepancy is the reason why, in our experimental and practical work, physiology and pathology are such close neighbors. But in trying the new way outlined above we may be able to separate this close relationship a little even if it be only by 0.1 mm.! In the routine way of practice nowadays, this neighborliness of physiology and pathology is obliterated entirely—everything is pathology.

Systemic Factors.—Besides the injuries to the tissues inflicted by mechanical influences, many other potential dangers should be taken into consideration and, if possible, eliminated or reduced before an orthodontic treatment is started.

This is not the place to discuss the many possible systemic factors which may be of great significance in causing failures during active treatment nor those responsible for some unfavorable aftereffects. This topic is dealt with by Pottenger[35] and others and quite comprehensively also by Becks,[3] pointing out the good and poor risks of an orthodontic treatment. In this paper Becks states (p. 622): "Dentistry in the past, and orthodontics in particular, has been a profession which dealt only with engineering problems in the large field of biologic science. Let us endeavor to change this commonly accepted point of view and try to arrive at a high standard of dental education and professional endeavor guided by a sound dentomedical approach." It is the belief of all research men in the medical field who are interested in our speciality that "orthodontia is not a dental problem alone; it is also a medical problem." The lack of cooperation between medical men and orthodontists lead Pottenger[35] to say: "Such cooperation should materially lessen the criticism that orthodontic procedures fail in so many individuals." "There is something rotten in the realm of orthodontics" (to paraphrase Shakespeare), otherwise we would not find so many similar remarks in the recent orthodontic and medical literature.

The immediate result should not count too much; more consideration should be given to the long afterresults. Of course the beneficial effect of such an attitude can only be obtained by close collaboration of orthodontists and the general dental practitioners who are called upon to treat the paradentoses which may have had and often do have the orthodontic treatments among their predisposing causes.

One more final remark: Every branch of human enterprise that strives for progress and evolution stands upon the foundation of theory and research. This applies especially to medicine and its important branch, dentistry. In this country of unlimited wealth, many philanthropists have dispensed many millions to establish schools and research laboratories for the advancement of

dentistry. This money and time and work spent in these laboratories did not fall on barren ground. Everywhere the marvelous results of this research are highly appreciated and put into practice by the profession and gratefully accepted by the laity. But from this generally accepted attitude, that research should be the basis for practice, orthodontics seems to be a glorious exception. Little attention is paid to the results of research, to the demonstration of what is happening invisibly below the mucous membrane; most of the interest is still concentrated on mechanics alone. Orthodontics is now confronted with a chaos of individual philosophies not based on research. Unless that is changed, histologic research will have little chance. That research undoubtedly has direct relationship to, and yields valuable information for, the practice, finds expression again in the concluding sentence of Gottlieb's latest contribution:[12] "The result of today's research is destined to be an integral part of the practitioner's work of tomorrow."

With the same idea in mind, Orban wrote the concluding sentence in the preface of his latest book:[33a] "We dedicate this book to those who recognize that clinical procedure is based on the knowledge of normal structure."

One does not realize strongly enough that no force is lost in the mouth and that even infinitesimally small forces produce enormous changes. The erroneous belief that a reliable unyielding anchorage can be established in the mouth is misleading and is the cause of many failures.

Orthodontists should discipline themselves to think not only in terms of mechanics but also in terms of biology. If this goal were attained, an approach to biologic treatment would result. The supreme law of medicine "Primum non nocere (first of all beware of inflicting damage)" would then be made the supreme rule in orthodontics also and would make it a real and equivalent partner of medicine.

SUMMARY

Through the stimuli of light forces, osteoclasts are mobilized after a very short time and attack the bone by uniform superficial lacunar resorption. The lighter these stimuli, the higher the number of our great helpers which remove the obstacles for the movement. They persist for at least four days after force discontinuation and continue to work toward the same goal. For these cells we propose the name "primary osteoclasts."

In the use of strong forces the periodontal membrane becomes crushed and its nourishment facilities cut down. As a consequence, undermining resorption sets in from various sides but not only in the direction of the intended movement. The resorption (loss) of bone is therefore not limited. The osteoclasts on the periosteal side appear already after twenty-four hours, possibly even earlier. For both these osteoclasts we propose the name "secondary osteoclasts." They persist until the affected bone, cementum, and the crushed tissues are eliminated.

In the use of strong forces, many vessels are ruptured on the traction side, forming hemorrhages. The corresponding impairment of the nourishment of the bone with the inevitable encroachment on the osteocytes, as well as the toxins originated by the decomposition of the red blood corpuscles, mobilize osteoclasts

to work on the traction side too. This contributes to the looseness of the teeth. For these osteoclasts originating under quite strange conditions we propose the name "tertiary osteoclasts."

On the pressure side, on account of impaired nourishment by the total compression and thrombosis of the vessels, the osteocytes and cementum corpuscles disintegrate and finally disappear. Structures deprived of their living elements are "dead" tissues and sooner or later Nature has to dispose of them.

To avoid all these occurrences we have so far only one means, that is, to avoid a too great or too long protracted compression of the periodontal membrane.

The development of cementum resorptions depends on the duration, or rather on the intensity, of the force.

A force application of 240 or 360 grams as used in these experiments is much too strong; with such forces no physiologic reaction can be brought about any more.

The pathologic changes observed after several weeks of movement in the apical third of the pulp depend on the great shifting of the apex. In avoiding this the damages to the pulp will be eliminated correspondingly. All the damages to the periodontal membrane, bone, cementum, and pulp could probably be avoided by the use of forces (stimuli) of only short duration (one day or one night) followed by rest periods of even longer duration (one or even two to three days). *The force may not be applied again as long as the once created osteoclasts continue to work* (four days). Thus, a continuous superficial osteoclastic activity could be brought about working only in the direction of the intended movement. Such a procedure may prove not only the safest but also the quickest way; at the same time an approach to biologic movement may be achieved. But all this is only feasible with partly removable appliances or appliances still to be designed for different movements. The objection, that we then depend entirely on the patient's cooperation, is not a sound argument; in the use of any kind of appliance we have to rely on the patient's cooperation. Osteophyte formation is usually quite poor in man; their formation needs time. To assure or favor such a formation, slow progress of the movement is the only answer. Osteophytes are Nature's only preventive measure against too great but not against too quick, a thinning of the bone.

Conditions and circumstances were shown which may prove the potential cause for future paradentosis.

It was verified that osteoid bone offers greater resistance to resorptions than well-calcified bone. In the use of strong forces no osteoid forms on the traction side.

The stronger the force used, the greater the probability for a quick and forceful relapse. One reason for these relapses may also be the too wide periodontal space at the traction side caused by the nondevelopment of osteoid and still enhanced by the action of osteoclasts.

Several statements made already on previous occasions were repeated in this essay in order to give a synopsis of the whole situation, which is not yet too clear.

CONCLUSIONS

The histologic evidence in this essay would seem to prove these points: (1) That the application of light forces is correct and preferable in orthodontics. (2) Comparison of the hours of actual force influence in continuous treatment with the treatment at night only show that there is a decided advantage in the latter. (3) Our work should be performed so slowly as to give Nature ample time for the compensatory formation of osteophytes. For the same reason the osteoid on the traction side is laid down in an even layer and is of greater thickness. By the reduction of the width of the periodontal membrane and on account of the greater resistance of osteoid to resorption, the extent of an always possible relapse is greatly limited. (4) Only light forces are able to produce an abundance of the *primary osteoclasts* which alone can be considered our real helpers. They alone work without creating too great damage, if any at all. These primary osteoclasts are the principal factor in bringing about all the marvelous and revolutionary changes without clouding the prospects for the future.

(*To be continued*)

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122 EAST 16TH STREET

Editorial

The Oppenheim Research

Tissue change incident to tooth movement was an entirely speculative matter until Dr. Albin Oppenheim of Vienna made his epoch-making contribution to orthodontics in 1911. The Oppenheim research, which was performed at that time upon monkeys, made possible certain deductions pertaining to the movement of teeth which added much to the general advancement of orthodontics. This work was published in the "Angle Orthodontist," Volume 3, Numbers 2 and 3, by the Alumni Society of the Angle School of Orthodontia, and since that time has been widely quoted in orthodontic literature, both in textbooks and in manuscripts in scientific periodicals.

As a result of the research, Dr. Oppenheim was the first to point out the necessity for slow, gentle pressure to be applied in the movement of teeth, and he submitted scientific proof to support that observation. His work turned orthodontic attention entirely away from the twisted ligature or grass-line type of orthodontics being practiced at that time, because it revealed that such methods of moving teeth were entirely too violent to be physiologic in result.

Now, thirty-three years subsequent to that contribution, in this issue of the JOURNAL, appears Dr. Oppenheim's latest contribution to this subject in which he presents striking new evidence that the bone does not respond to strong mechanical forces in such a way as to be of advantage in orthodontic treatment. Dr. Oppenheim feels that orthodontists are still using entirely too much mechanical energy in the movement of teeth and he sets about to prove it.

The new research, which was completed in Los Angeles in connection with the University of Southern California, is published in two parts, the second part of which will appear in the next issue of the JOURNAL.

A study of these articles reveals that osteocytes (bone cells), in bone which is subjected to too much pressure, die and disappear because their nutrition is reduced or cut off by the compression of the blood vessels. It is shown that bone containing dead cells must be eliminated, and this results in the loss of important supporting structure which may never be restored after orthodontic treatment.

The author's experiments were made on monkeys; subsequent to this, the author and some friends and co-workers (Orban, Sicher, Weinmann) re-examined their work on dogs and human material, which, when subjected to similar strong forces, showed the same destruction of bone cells. Oppenheim shows that, under strong forces, the osteoclasts cannot work directly on the

surface in direct line with the pressure. It becomes necessary for them to work around the block of dead bone where they are needed the most, and just ahead of where the pressure is applied. When this obstruction is removed, according to him, the tooth goes right ahead but not necessarily in the direction which is desired because the progress and direction of the undermining resorption, cannot be guided or checked. It cannot be denied that, with strong forces also, results may be secured in the end, but at a very high risk and with so much more tissue mutilation and trauma than is necessary.

Another startling fact is that where vigorous forces have been used, osteoclasts appear not only on the periodontal side (undermining resorption) but also on the periosteal side of the bone against which the pressure is applied as well as on the traction side. On the traction side the violent movement tears and blocks the blood vessels and capillaries, resulting in blood hemorrhages. The red blood corpuscles disintegrate, producing toxins, and the osteoclasts which come into being now attack the bone on the traction side too. To put it in clinical language, the tooth sockets become wide and large, and the teeth are loose and sore, for we now have a pathologic condition on the traction side also, in contrast to a physiologic proposition.

Another fact is pointed out that is plainly just as important as the one just given, and that is that bone cells are choked by too much pressure. Oppenheim has proved by his experiments that primary osteoclasts brought into being by light stimuli remain active for at least four days after orthodontic apparatus is removed. He claims, therefore, that teeth may be moved physiologically and satisfactorily by using appliances only at night with no apparatus applied in the day. This idea is not entirely new because some orthodontists have been doing this for some time; they having discovered its efficiency by clinical observation alone. Particularly is this true of the Baker anchorage.

The theoretical, first part of Dr. Oppenheim's paper is supplemented and supported by a practical, second part in which cases are shown treated by orthodontists, following the principles dictated by the findings in the first part. It is shown, though the time of treatment as a whole is prolonged, that the time of actual force application is highly reduced, which means considerably less strain on the tissues and, therefore, better chances for repair and restoration.

You should read the Oppenheim manuscript and research report because it adds much to the contribution made by the same author on the same subject thirty-three years ago. It will also serve the purpose of inspiring you to harder work and to the realization that research is contributing much to the fundamental groundwork of orthodontics and making for a brighter future for the subject. This is another of the very important contributions to orthodontic advancement, in which the scientific information revealed can be applied quickly to everyday clinical practice.

H. C. P.

Chicago Meeting of the American Association of Orthodontists

Under the energetic leadership of Dr. James A. Burrill and with the help of his efficient committees, the Forty-Second Annual Meeting of the American Association of Orthodontists was held at the Edgewater Beach Hotel, Chicago, Illinois, April 25, 26, and 27.

The meeting "got away" to a lively start when the annual stag dinner and "get-together" was staged at 7 o'clock on Monday evening. The stag had many of the earmarks of "old home week" inasmuch as there were so many greetings to be extended after two years.

On account of the dislocated conditions that exist at this time, this was the only meeting held within the last two years. Accordingly, there was the largest registration of any gathering within the history of the Association, totalling six hundred and sixty-seven, which included the wives of some of the members.

The meeting was interesting and was efficiently handled, everything going off according to schedule. Enthusiasm and interest could be noted everywhere. The Clinics were unusually numerous and interesting. One enthusiastic group, featuring extraction and no extraction cases, brought a large and complete exhibit all the way from Seattle, Washington.

Probably the most spectacular event of the meeting from the standpoint of member interest was the extraction panel, in which was discussed "The Place of Extraction in Orthodontic Procedure." This discussion occupied the entire forenoon of Wednesday, April 26. The panel was under the direction of George W. Hahn of Berkeley, California, and those who spoke formally upon the subject were: Charles Tweed of Tucson, Milo Hellman of New York, George W. Grieve of Toronto, and Allan G. Brodie of Chicago. The men invited to participate in the discussion obviously were chosen because each is an authority in his field and is interested in the progress of orthodontics. Following the program there was an informal discussion between the members of the panel which consisted largely of questions and answers.

The formal program of the meeting was one of the best which has been held and was well attended by attentive audiences.

The President's Ball and Annual Banquet was held on Wednesday evening. Something new in the way of entertainment consisted of a routine by professional entertainers who, by means of a loud speaker, and accompanied by an organ, kept up a running fire of quips during the entire evening in an orthodontic lingo which only an orthodontist could understand. This proved to be a happy choice on the part of the entertainment committee, as the guests found it very amusing.

The American Board of Orthodontics reported thirty-one applications, the largest number to be acted upon at one time in the board's history. The persistent demand for the certification of the American Board proves the prediction of the late Dr. Ketcham, its founder, that the board would serve as an important medium, within a very few years, to maintain a higher standard of orthodontic practice throughout America.

According to the results of the election, those who will lead the organization for the coming year are:

Archie B. Brusse, Denver, Colorado, President.
Earl G. Jones, Columbus, Ohio, President-Elect.
William G. Sheffer, San Jose, California, Vice-President.
Max E. Ernst, St. Paul, Minnesota, Secretary-Treasurer.
Richard A. Smith, Evanston, Illinois, Librarian.

Further information, committee reports, awards, etc., will be reported in the JOURNAL at a subsequent date. That the meeting was a great success was attested to by all who attended.

H. C. P.

Honor Roll of Active Members
American Association of Orthodontists
Serving in the Armed Forces

Dr. Herman Adelstein
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Dr. C. A. Allenburger
New Orleans, La.
Dr. W. R. Alstadt
Little Rock, Ark.
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Dr. Ernest E. Palmatary
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Dr. J. D. Peak
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American Association of Orthodontists
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(Continued)

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Dr. Joe Tennyson Reece
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Dr. Paul V. Reid
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Regular Army Service Members

Col. Harry Deiber
Col. Neal Harper
Col. Wm. H. Siefert
Col. Richard F. Thompson
Col. L. B. Wright

There may be members in the Service whose names do not appear in the above list. These members should notify the secretary at once so that their names may be included.

Max E. Ernst, Secretary, American Association of Orthodontists, 1250 Lowry Medical Arts Bldg., St. Paul, Minn.

Department of Orthodontic Abstracts and Reviews

Edited by

DR. J. A. SALZMANN, NEW YORK CITY

All communications concerning further information about abstracted material and the acceptance of articles or books for consideration in this department should be addressed to Dr. J. A. Salzmann, 654 Madison Avenue, New York City

Oral Histology and Embryology: Edited by Balint Orban, Foundation for Dental Research of the Chicago College of Dental Surgery, School of Dentistry, Loyola University. With 262 text illustrations including four color plates, pp. 342, price $6.50, St. Louis, The C. V. Mosby Company, 1944.

A new plan was followed in the compilation of this volume. Chapters were prepared by recognized authorities in their specific subjects. Each author's manuscript was then submitted to all other contributors for discussion. Some of the contributors did not write a chapter but aided the effort by their remarks and criticism. It speaks well for dentistry to know that fifteen of the nineteen contributors are holders of the D.D.S. degree, and four others have been long associated with dental teaching and research.

Such a collection of authorities would have been hard to find in dentistry twenty years ago. Included among the contributors are Aisenberg, Bernier, Bevelander, Bodecker, Cheyne, Diamond, Gottlieb, Kerr, Kitchin, Kotanyi, Mueller, Nuckolls, Orban, Schour, Sicher, Skillen, Weinmann, Worman, and Zander.

The chapter on the "Development of the Face and Oral Cavity" is exceptionally well illustrated and should prove of definite value to the student. In the part of the text dealing with "Development and Growth of Teeth," the work of Schour and Massler is largely followed. The various structures of the teeth and the soft structures surrounding the teeth are illustrated in detail.

A valuable chapter will be found on the development and growth of the maxilla and mandible, based largely on the work of Sicher and Tandler. Here the contributions of Brodie have also been given due consideration. Shedding of the deciduous teeth and tooth eruption as may be expected, are given full consideration. The photomicrographs presented surpass most of those found in dental or medical books that have come to the attention of this reviewer.

This is a book in which the leading contributors on oral histology and embryology have participated and one of which the dental profession may well feel proud. It can and will be used not only by the student but by the practitioner of dentistry as well. The book has an excellent index which will be especially useful in reference work.

Oral Pathology. A Histological, Roentgenological, and Clinical Study of the Diseases of the Teeth, Jaws and Mouth: By Kurt H. Thoma, D.M.D., Professor of Oral Surgery and Brackett Professor of Oral Pathology, Harvard University; Oral Surgeon and Chief of Dental Service, Massachusetts General Hospital; Oral Surgeon to Brooks Hospital; Dental Surgeon to Dental Department and Consultant in Oral Surgery to Tumor Department, the Boston Dispensary, and Joseph H. Pratt, Diagnostic Clinic, Consulting Oral Surgeon, New England Baptist Hospital; Consulting Surgeon, Beth Israel Hospital. Second edition, 1328 illustrations including 128 in color, pp. 1328, price $15, St. Louis, The C. V. Mosby Company, 1944.

In this, the second edition of Thoma's book, a number of the rarer diseases originally omitted from the first edition have been included, and many new illustrations have been added while others have been replaced. Weisberger and Goldman have aided the author in bringing the chapters on mouth and periodontal diseases up to date.

Part I includes chapters on hereditary influences in anomalies of the teeth and jaws, and endocrine and nutritional influences in the development of the teeth and jaws. *Part II* deals with developmental anomalies of the teeth, and *Part III* with the developmental anomalies of the head. *Part IV* treats of the functional changes of the teeth, and includes physiology, pathology, environmental pathology, and an interesting chapter on the pathologic effects of dental restorations. *Part V* presents traumatic injuries of the teeth, and *Part VI* includes dental caries, pulp involvement, and dentoalveolar abscesses. Parts are included also on periodontal diseases, extension of odontogenic infections and diseases of the jaws.

An excellent chapter is that on "Tumors of the Jaws and Diseases of the Oral Mucosa." Diseases of the lips and tongue are presented in great detail. A wealth of references and a detailed index are included. This is an outstanding and authoritative contribution to dental literature.

Traumatic Injuries of Facial Bones: By John B. Erich, M.S., D.D.S., M.D., Consultant in Laryngology, Oral and Plastic Surgery at the Mayo Clinic, Assistant Professor of Plastic Surgery, The Mayo Foundation for Medical Education and Research, Graduate School, University of Minnesota, and Louie T. Austin, D.D.S., F.A.C.D., Head of Section on Dental Surgery at the Mayo Clinic, Associate Professor of Dental Surgery, The Mayo Foundation for Medical Education and Research, Graduate School, University of Minnesota. In Collaboration with Bureau of Medicine and Surgery, U. S. Navy. Pp. 600 with 333 illustrations, Price $6.00, Philadelphia and London, W. B. Saunders Company, 1944.

Anyone who has ever questioned the role of the orthodontist in modern warfare should be referred to this volume. Here he will find almost 600 pages of material which testify to the valuable service which the orthodontist is capable of rendering in the treatment of combat injuries of facial bones. The value of the knowledge which the orthodontist possesses is an indispensable factor in such procedures as the construction of dental splints used in stabilizing traumatized facial bones and in intraoral, extraoral methods of wiring. The con-

struction of anchor bands, the attachment of arch bars to partially edentulous jaws, and circumferential wiring of the mandible all involve procedures familiar to the orthodontist. The orthodontist is working in his own field also when he is constructing plaster headcasts, orthodontic jackscrews, traction and fixation appliances.

It should be kept in mind that the restoration of fractured and mutilated jawbones requires a thorough knowledge of dental occlusion. Who, more than the orthodontist, understands occlusion of the teeth and its vagaries? All of the aforementioned have been included in this book which is beautifully illustrated with color plates and black and white drawings.

The use of extraoral skeletal fixation apparatus has not done away with intraoral splinting. This book has been divided not only into chapters but also into individual problems, each of which deals with a specific type of injury of the facial bones. A detailed account in brief form is presented of the methods of treatment which have been most effective and appropriate.

This is a volume which has been prepared primarily as a handbook to enable the practitioner to obtain essential information concerning specific problems.

Erich, who is both a physician and dentist, and Austin, a dentist, bring to this field a sound knowledge of the mechanics and methods employed in constructing the various dental and orthodontic appliances. One can easily agree with Rear Admiral Ross T. MacIntyre when he expresses the grateful appreciation of the medical department of the Navy for this work. This book can be heartily recommended to those interested in this field.

Atlas of the Mouth and Adjacent Parts in Health and Disease: By M. Massler, D.D.S., M.S., Director of the Child Research Clinic, Assistant Professor of Histology and Lecturer in Stomatology (College of Medicine), and I. Schour, D.D.S., Ph.D., D.Sc., Professor of Histology and Head of the Department of Histology, University of Illinois College of Dentistry. Drawings by Carl T. Linden, 180 illustrations (19 in color), Price $2.50, Published by the Bureau of Public Relations, Council on Dental Health of the American Dental Association, 222 East Superior Street, Chicago 11.

There has for some time been a need for an atlas of the oral structures in health and disease that was little recognized until the publication of this book. Now that it is available its usefulness and interest should rapidly become apparent.

As the name implies, it is an atlas and not a textbook. For that reason it has especial value in several different fields of education. It may be used and will be very useful in conjunction with dental texts, almost all of which are deficient in the type of illustration found here. Its usefulness to the dentist is not, however, limited to his undergraduate days. The practitioner will find it helpful in practice as a reference in identifying conditions rarely seen, also in visual education for his patients.

It will be useful as an adjunct in courses of dental lectures and demonstrations for medical students and nurses, even though much of it may seem to be of dental interest only. The drawings showing the various anatomic rela-

tionships are of medical as well as dental interest and serve to attract the attention (not too easily gained) of the medical student to other illustrations more strictly dental.

The common diseases of the teeth and their supporting structures are well shown. The dentist may, however, be more interested in the illustrations, many in color, depicting the common lesions of the buccal mucosa and lips, and also of the tongue. The blood and nerve supply of the jaws and head are well shown.

The illustrations are accompanied by brief descriptions, making this, in a way, a reference book in digest form. Brief but adequate references are given.

The illustrations throughout exhibit a combination of artistic talent and knowledge of dental anatomic detail that is rare indeed. The authors are most fortunate in having had the services of Mr. Linden for this all-important task. The color plates are particularly lifelike, something which can seldom be said of artists' renderings of dental and oral lesions. The lettering by Miss Marion Mason adds to the attractiveness of the illustrations.

Attention is called to the low price of this book, made possible by its publication on an entirely nonprofit basis.

It is a splendid reference book for the dentist.

John Oppie McCall

Dental Practice Management: By Wm. H. O. McGehee, D.D.S., M.D., Late Professor of Dental Practice Management, Georgetown University; Professor of Operative Dentistry and Secretary, New York University College of Dentistry, and Alfred S. Walker, D.D.S., Professor Emeritus of Pulp Canal Therapy and Special Lecturer on Practice Management, New York University College of Dentistry, former member of Board of Dental Examiners, State of New York. Pp. 285, Price $3.00, Chicago, The Year Book Publishers, Inc.

The distinguished authors of this book have given the profession something of unique value in its field. Books on practice management are not a rarity but it is unusual to find so much of practical value, interest, and applicability in a book on this subject.

Practice management is probably the weakest point in the equipment of the embryo dentist. Little wonder that the dentist just out of college feels the need for association with an older practitioner so that he may learn by day-to-day observation just how a successful practice is carried on. Even there he will find some questions unanswered, for his preceptor will assume his gradual absorption of the various niceties that enter into the daily routine of the office.

The authors of this book have anticipated all of these points and have given the answers to the problems clearly and graphically. The advice given is obviously not founded on theory, and the solution of many of the problems cited is given in the form of description of actual incidents in which the aid of one or the other of the authors has been sought.

One of the most important bits of advice in the book is that which urges the young dentist, or even the older one, to analyze himself as to his qualifications, desires, and personal qualities. The young dentist is urged to set up at the outset of his career definite objectives as to the type of practice he desires

to develop. For the older practitioner who has not succeeded as yet, this introspection supplemented by frank conference with a trusted friend or teacher may be the turning point.

The book is in three main sections: Ethics as applied to dental practice; Economics, the business management of professional practice; Jurisprudence, the law as applied to dental practice. Each section is well developed and each makes its own contribution to the subject as a whole. The book should be not only read by, but should be the constant companion of, the young practitioner.

J. O. McCall

The Premature Infant: By Morris Gleich, M.D., *Arch. Pediat.* **59**: No. 1, January, 1943.

A premature infant is born with an inadequate supply of necessary minerals because a large part of these is given to the fetus in the last two months of intrauterine life. The rapid growth of many of these infants, their insufficient food intake, and their inadequate supply of minerals at birth make them an easy prey to rickets, tetany, and anemia. Calcium and phosphorus are necessary ingredients of normal bone and tooth structures. Their metabolism is favored by the intake of vitamin D. The latter liberates phosphatase. This enzyme splits organic phosphorus into inorganic phosphorus. The inorganic phosphorus combines with calcium to form bone. Milk contains sufficient amounts of calcium and phosphorus for the proper growth and development of the premature infant. With a sufficient vitamin D intake and a normal parathyroid, additions of calcium and phosphorus to the dietary are unnecessary.

Mouth.—Since the buccal mucous membrane of the infant's mouth is very delicate, it is inadvisable to wash it. Nurses and parents should always be on the lookout for thrush.

Harelip and Cleft Palate.—While harelip is usually operated on in a vigorous infant in the first two weeks of life, it is wise and essential that operation upon a premature infant be deferred until the baby is able to withstand this procedure. As a rule, operation on cleft palate is performed some time between the ages of 18 months and 5 years. The longer one waits, the greater the chances for speech defects.

Preoperative and Postoperative Precautions.—In any operative procedure on a premature infant it is wise to build up the infant. In the event of a low blood prothrombin content, we advise the use of vitamin K, by mouth, 1 to 2 c.c. every four hours for at least four to six doses for at least 24 hours before operating.

Following an operation, where the infant is still under the influence of the anesthetic, place in a Trendelenburg position to avoid the aspiration of mucus into the lungs, preventing pneumonia. Give food in smaller amounts, diluted, and at longer intervals. To prevent dehydration, clyses may be given every four to eight hours for 24 to 48 hours after an operation, because the fluid intake by mouth is reduced.

News and Notes

Harvard School of Dental Medicine*

CLINICAL DENTISTRY

Dr. Alfred LeRoy Johnson, Professor of Clinical Dentistry, who has been for some months Executive Officer of the new Harvard School of Dental Medicine, has been named (April 1) Administrative Officer of the School and Associate Dean of the Faculty of Medicine. With the graduation of the last class of the Harvard Dental School (*Bulletin*, March 31), dental education and research in the University have become the province of the new School.

Dr. Johnson graduated from Tufts Dental School in 1904. After practicing as a dentist, he served as professor of orthodontics at Tufts, University of Michigan, and the University of Pennsylvania. Since 1931 he has been a research associate in experimental genetics at the Cornell Medical School. He came to Harvard in 1942 as Professor of Clinical Dentistry. He is the author of *Basic Principles of Orthodontics*, *The Constitutional Factor in Skull Form and Dental Occlusion*, and other works in this field; and co-author, with Dr. C. R. Stockard, of *Constitutional Basis of Form and Behavior*.

The new School will have a small number of carefully selected students each year who, during the first two years, will take a course identical with that given for students in the Medical School. At the end of four years they will receive the degree of D.D.S. If they desire to attend the Medical School for an additional year, and qualify, they will also have the opportunity to receive the M.D. degree.

The new School is one of the institutions designated by the Navy for training men for its Dental Corps. Eight such men have already been assigned to it in addition to its civilian students.

THE NEW PROGRAM

Under Dr. Johnson's direction, the School of Dental Medicine now takes all responsibility for Harvard's dental program, supported by an endowment of nearly $4,000,000. On the day following the announcement of his appointment the new administrative head† outlined the purposes and plans of the School to a large meeting of Alumni in the dental school building. Speaking with clarity and directness, he set forth a threefold purpose:

1. To train men for the practice of dentistry.
2. To stimulate interest in research and teaching.
3. To offer opportunity for graduate experience in special fields.

. . . Its philosophy is to create a study center permeated by an atmosphere of learning that will encourage a student to broaden and deepen his understanding of the basic sciences of health as well as to develop his technical skills; to make him realize that education does not stop when he receives his degree but is a life-long and an age-long process.

Such a philosophy implies continuous emphasis on the practical value of the scientific method of thought. This is not a revolutionary step. It is plain common sense. For in the final analysis science is responsible for all that is truly practical in dentistry. The unfortunate tendency to contrast the scientific approach with the practical approach has compromised both and to a certain extent at least has been a hindrance to progress. . . . If our present program fails to train dentists with a scientific interest in their work, the program will be changed, and always with this end in view.

*Reprinted from the *Harvard Alumni Bulletin* of April 15, 1944.

†Dean LeRoy M. S. Miner continues at Harvard as Professor of Oral Surgery in the Faculty of Medicine.

RELATION TO MEDICINE

Dr. Johnson then answered squarely a number of criticisms which have been freely leveled in certain dental quarters against the conception of the new School. Taking first the question whether, as a result of affiliations with Medicine and Public Health, dentistry would lose its autonomy, as "ghost-chasing," he said:

There is nothing revolutionary in such [affiliation]. In adopting it we merely have conformed to the natural trend of events. . . . The identity of the School of Dental Medicine will be maintained, and there are absolutely no grounds for believing otherwise. The School of Public Health, the Medical School, the School of Dental Medicine, each has its task and function. Their respective fields are becoming more clearly defined. And it is the belief of those now at work on this definition that the integration of certain aspects of each with one and, in some instances, with both of the others, will increase the effectiveness of each individual school.

QUALITY STANDARDS

Turning to the question, "Why are the entering classes of the new School to be limited to fifteen students?" he pointed out that the course of the first two years of the new School would be identical with that of the course in medicine; that the science laboratories of the Medical School are equipped to accommodate only 125 students and that the admission of fifteen students training for dentistry would leave only 110 places for medical students. An increase in the number of dental medicine students beyond fifteen would necessitate expansion of facilities and teaching staff in the basic science departments which is not practicable at this time.

. . . To some the size of this class may seem small and capital is being made of the fact that Harvard will turn out fewer dentists at a time when more are needed. There is no doubt that dental service today is not effective as a social service. The conventional type of dental education has demonstrated its inability to meet the situation. And the same is true of medical education. At this time I shall not discuss the comparative merits of plans proposed for the extension of dental service. Our position is that this problem of scarcity involves quality of professional personnel as much as it does quantity. We, of this school, believe that the greatest service Harvard can render is to place all emphasis on quality and understanding even at the expense of quantity.

In conclusion, Dr. Johnson reassured doubters as to the intention of the new School to carry on clinical work. This, he said, would be done, and both part-time and full-time men will be represented on the clinical staff of the School.

Letter to Deans of Dental Schools

Taken from a letter sent out to deans of dental schools on May 21 by C. Willard Camalier, Chairman of the War Service Committee, the following quotation is of interest:

The Surgeon General's Office has announced that Army dental students under the AST Program, *for the June, 1944, class only*, totaling approximately 250, will be honorably discharged upon completion of their studies, as the quota of officers in the Army has been reached for the present.

These men, however, will be expected to apply for commissions in the Navy as that agency is endeavoring to commission about 200 officers per month for the balance of the calendar year. A few may be commissioned in the Veterans' Administration and some could be utilized as interns in the U. S. Public Health Service, in approved civilian hospitals, or in clinics such as the Eastman Dental Dispensary, Rochester, N. Y., Forsyth Dental Infirmary, Boston, Mass., the Murry and Leonie Guggenheim Dental Clinic, New York City, or as full-time teachers in schools of dentistry.

It is not expected that these graduates will attempt to practice in a civilian capacity during this emergency unless they are physically disqualified by the Navy or have applied to and been accepted as full-time teachers by a school of dentistry or by one of the agencies mentioned above.

It is suggested that the directors of these institutions contact, for available interns, the schools graduating these men, and that the deans themselves consider others for full-time teaching positions.

Central Section of American Association of Orthodontists

The Central Section of the American Association of Orthodontists met for a business and luncheon meeting on May 27, 1944, at the Edgewater Beach Hotel. Dr. Charles R. Baker, president, was in charge of the meeting. The following were elected to membership in the Central and American Associations:

Asher, Dr. S.	4753 Broadway, Chicago 40, Illinois
Berman, Dr. M. C.	55 E. Washington St., Chicago 2, Illinois
Burrill, Dr. D. Y.	25 E. Washington St., Chicago 2, Illinois
Delbridge, Dr. H. N.	216 Strong Bldg., Beloit, Wisconsin
Dreiling, Dr. F. J.	835 Argyle Bldg., Kansas City, Missouri
Foyle, Dr. F. A.	6315 Brookside Plaza, Kansas City, Missouri
Johnson, Dr. O. E.	753 E. 70th Street, Chicago, Illinois
Kloehn, Dr. S. J.	515 Zuelke Bldg., Appleton, Wisconsin
Kral, Dr. Ione J.	137 N. Marion St., Oak Park, Illinois
Milliette, Dr. G. T.	1st Nat'l Bank Bldg., Milwaukee, Wisconsin
Mueller, Dr. H. H.	418 Exchange Bldg., LaCrosse, Wisconsin
Schwartz, Dr. H.	55 E. Washington St., Chicago, Illinois
Winter, Dr. W. W.	769 Citizens Bldg., Decatur, Illinois

Officers for the coming year were elected as follows:

President - - - - - - - - Dr. A. C. Rohde, 324 E. Washington St., Milwaukee, Wis.
President Elect - - - - - - - Dr. C. S. Foster, 803 Dows Bldg., Cedar Rapids, Iowa
Vice-President - - - - - - - Dr. R. G. Bengston, 25 E. Washington St., Chicago, Ill.
Secretary-Treasurer - - - - - - Dr. L. B. Higley, 705 S. Summit St., Iowa City, Iowa
Representative to Board of
Directors of the A. A. O. - - - - Dr. O. W. Brandhorst, 4500 Olive St., St. Louis, Mo.
Alternate to Board of
Directors of the A. A. O. - - - - Dr. J. A. Burrill, 25 E. Washington St., Chicago, Ill.

The Central Section contributed the second Angle Memorial Lecture to the program of the American Association meeting which was in session.

—L. B. Higley, Secretary.

Commissioning of Women Dentists

Women dentists, who will serve alongside the men of the Dental Corps of the U. S. Navy, are to be commissioned through the WAVES, according to an announcement received by the Liaison Office of the American Dental Association, from the Navy Department.

Under the new ruling which will enable women dentists to serve in the same capacity as men, the qualifications are the same as for men dentists except that duty will be within the Continental limits; the applicant may not be married to an officer in the Navy, and may not have children under 18 years of age.

Further information may be obtained at any office of Naval Officer Procurement or at the Bureau of Naval Personnel, Washington, D. C.

At the same time, Captain Joseph Tartre, U.S.N., D.C., announced at Great Lakes Naval Training Center that Dr. Sara G. Krout of Evanston, Illinois, has been commissioned a full lieutenant in the Dental Corps of the U. S. Naval Reserve, through the WAVES, and reports for duty at Great Lakes on June 1. Dr. Krout becomes the first woman to be appointed to such duty in this area. She will treat regular Navy personnel as well as WAVES who are stationed at Great Lakes.

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Central Section of the American Association of Orthodontists

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*The Journal will make changes or additions to the above list when notified by the secretary-treasurer of the various societies. In the event societies desire more complete publication of the names of officers, this will be done upon receipt of the names from the secretary-treasurer.

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*The Journal will publish the names of the president and secretary-treasurer of foreign orthodontic societies if the information is sent direct to the editor, 8022 Forsythe, St. Louis 5, Mo., U. S. A.

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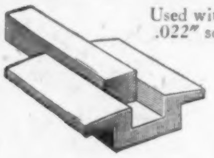

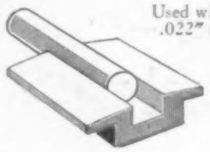

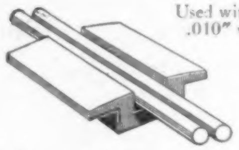

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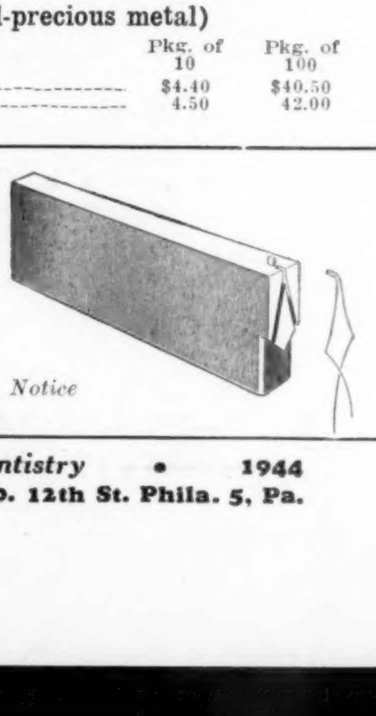

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Mayo Clinic Number

From the Sections on (1) Dental Surgery, (2) Otolaryngology and Rhinology, and (3) The Division of Medicine, The Mayo Clinic, Rochester, Minn.

PENICILLIN IN THE TREATMENT OF CELLULITIS OF THE MOUTH

WALLACE E. HERRELL, M.D.,* AND DONALD R. NICHOLS, M.D., ROCHESTER, MINN.

CELLULITIS of the floor of the mouth is considered a virulent and often a serious type of inflammation. If the infection is extensive, the process may terminate fatally. It is difficult to obtain accurate figures as to the mortality rate in cases of this infection. According to Taffel,[1] the mortality rate in many series of cases is somewhere between 25 and 54 per cent. Williams and Guralnick[2] reported twenty cases seen at the Boston City Hospital from June, 1939, to June, 1942. In this series of cases, the mortality rate was 10 per cent. However, the mortality rate in a previous series of thirty-one patients reported by the same authors was 54 per cent. In cases in which patients recover from extensive cellulitis of the floor of the mouth, the convalescence is usually slow and often stormy. It is generally agreed that dental sepsis is the initiating factor in a high percentage of these cases. An incidence of dental sepsis as high as 90 per cent has been reported as the initial lesion preceding the development of the cellulitis.

Extensive cellulitis occasionally may follow abrasions of the cheek or mouth. Such abrasions may include bites, gunshot wounds, and other forms of trauma. Other conditions which may be found to be the origin of the infection include peritonsillitis, otitis, ulcers of the lip, and so forth. The cellulitis may develop secondary to the extraction of septic teeth although the infection may follow dental sepsis in which extraction has not been performed. It appears that septic molar teeth are the most frequent sources of infection. Various pathogenic organisms have been found in association with the cellulitis. The organisms isolated include streptococci, and often there may be associated staphylococci, Vincent's spirillum, and, in some instances, gas-producing organisms. Micro-aerophilic streptococci may also be isolated in some instances. It seems likely that these infections are, for the most part, polymicrobial rather than monomicrobial in type.

The inflammation may be of a so-called malignant type, which rapidly invades the surrounding tissues and involves the submaxillary space and tissues of the neck. The infection, on the other hand, may progress slowly and eventu-

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*Division of Medicine, Mayo Clinic.

ally form an abscess. As a rule, firm induration of the submaxillary and submental regions is characteristic. There also are extensive swelling and induration of the floor of the mouth, gums, and tongue. The tongue is pushed upward and backward. At times, the tongue is described as being "frozen" to the roof of the mouth. In most instances, an abscess occurs. The edema of the tongue and throat may interfere with breathing and, in the late stages of the infection, may produce asphyxia. The patients become acutely ill, and in some instances pathogenic organisms may invade the blood stream. There is often a chill, and, in practically all cases, fever of a varying degree is present. The clinical picture is usually that of marked toxemia. In some instances in which the inflammation is extensive, mediastinitis or thrombosis of the jugular vein may occur. Such infection of the floor of the mouth most frequently affects children and young adults; however, it occasionally may occur among older persons.

There is no uniformity of opinion as to the most satisfactory method of treating cellulitis of the floor of the mouth. It is uniformly agreed, however, that for the most part extensive infection in the floor of the mouth is difficult to treat satisfactorily. The local use of heat, roentgen therapy, and sulfonamide therapy, together with either minimal or radical surgical intervention, have been employed. All of these forms of treatment have proved of definite value. These measures no doubt have reduced the mortality rate of cellulitis of the floor of the mouth greatly. On the other hand, the convalescent period in most instances has been tedious and prolonged, especially in cases in which the inflammation has been extensive. It is interesting that some authors resort to nonsurgical treatment for the most part, whereas others advocate immediate radical surgical intervention. Taffel has reported forty-five cases of Ludwig's angina, in which forty-six operative procedures were performed in forty-three patients. Only two patients were treated without operation. There were only two deaths in this group of cases, which speaks fairly well for this form of management. As previously pointed out, the convalescence in most cases, even with surgical intervention, is rather slow.

Because of the rather highly effective antibacterial action of pencillin against the majority of the organisms isolated in these cases, we have used penicillin in a small group of cases of extensive cellulitis of the floor of the mouth. Penicillin was used only in cases in which the patients were desperately ill and in cases in which it was thought that penicillin therapy might be considered a life-saving procedure. Penicillin is an antibacterial agent which is produced by a certain strain of the mold, ***Penicillium notatum***. Alexander Fleming[3] was the first to note the bacteriostatic properties of the filtrate cultures of this mold. Later, the Oxford investigators (Chain, Florey, and others[4]) reported their observations on the chemotherapeutic activity of penicillin, and since then, extensive clinical study by investigators both in this country and in England has proved this antibacterial agent to be extremely effective in many different types of infection. Penicillin has been found to be most effective in the treatment of staphylococcic, gonococcic, pneumococcic, and hemolytic streptococcic infections. It is likewise quite effective against anaerobic organisms, as well as against the miro-aerophilic streptococci mentioned previously.

The administration of penicillin has not been found to be associated with any serious toxic effects. Penicillin can be administered intravenously or intramuscularly. It may also be used locally. It cannot be administered by mouth since the acid in the gastric contents destroys the potency of penicillin. Two methods of intravenous administration of penicillin are possible. It may be given by single intravenous injections every two to four hours. On the other hand, penicillin disappears rapidly from the blood stream; therefore, the administration in this manner is not the most desirable method. The other method of intravenous administration is by means of the continuous or nearly continuous intravenous drip. We[5, 6] prefer the continuous intravenous drip method because it maintains a relatively constant concentration of penicillin in the blood and because it is a simple and time-saving procedure. For the intravenous administration, penicillin is usually dissolved in physiologic salt solution, but it may be dissolved in a 5 per cent solution of dextrose in distilled water. A common method of administration is by repeated intramuscular injections. This is a satisfactory method of administering penicillin but requires frequent injections throughout the twenty-four hours and thus places considerable demand on medical personnel. It is possible that larger amounts of penicillin are necessary for treatment by means of the intramuscular administration than are required when the continuous intravenous method is used. The intermittent intramuscular administration, however, is definitely more desirable than the intermittent intravenous method.

We have used two preparations of penicillin for clinical purposes. One preparation is the sodium salt of penicillin and the other is the calcium salt. Both have been found to be effective and neither produces any significant toxic reactions. We have used penicillin in six cases of extensive cellulitis of the floor of the mouth. All of the patients were seriously ill. In two cases the cellulitis was complicated by the presence of septicemia. In one of these cases, the organism present in the blood was *Staphylococcus aureus,* and in the second, it was a hemolytic streptococcus. In the remaining four cases, the blood cultures were negative. The organism isolated in this group of cases was either a hemolytic streptococcus or *Staphylococcus aureus.* Sulfonamide therapy had been previously tried and found ineffective in all but one case. One of the patients nearly died before sufficient penicillin could be administered to control the infection. Two others showed evidence of overwhelming toxemia. In all of the cases, the patient had marked difficulty in swallowing. In several cases, the induration extended far down into the structures of the neck.

The penicillin was administered by the continuous intravenous drip method. The patients received from 32,000 to 40,000 Oxford units of penicillin in 2 liters of physiologic salt solution daily. Half of the daily dose in 1 liter of solution was attached to the intravenous apparatus morning and evening. The penicillin solution was administered at the rate of 30 to 35 drops per minute. There was marked clinical improvement in all of the cases, and the patients were able to swallow without difficulty within forty-eight to seventy-two hours. The temperatures, as a rule, subsided by lysis. The first sign of improvement was, as a rule, a decrease in the toxemia. In the two cases in which the cellulitis was complicated by septicemia, the blood cultures became negative within forty-eight

hours. In some instances the edema of the face disappeared dramatically; however, the induration subsided more slowly.

In one case, an abscess formed and ruptured spontaneously through the cheek. Drains were inserted to promote drainage in this case, but in no other instance was surgical intervention necessary. We do believe, however, that surgical drainage is indicated if large abscesses form.

The convalescence in these cases was usually rather short and uneventful. The administration of penicillin was continued until it was certain that the inflammation was well controlled. No other method of treatment, aside from local application of heat, was used in any of the cases after the administration of penicillin had been begun. As stated previously, five of the six patients had failed to respond to sulfonamide therapy.

Although the number of patients treated with penicillin for cellulitis of the floor of the mouth is small, it appears that penicillin therapy is an exceedingly effective means of treating this infection. The patients, although seriously ill, responded rather dramatically to penicillin therapy. There were no deaths, and the convalescence was relatively short and uneventful. In five of the six cases, the longest period of hospitalization was fifteen days. In the remaining case, the patient had an extensive infection complicated by septicemia and had to remain in the hospital twenty-eight days.

REPORT OF TWO CASES

A summary follows of two of the cases in which dental sepsis apparently preceded the onset of the cellulitis. In the first case, there was definite evidence of dental sepsis associated with the cellulitis, but no dental operation was performed. The sodium salt of penicillin was administered intravenously. In the second case, the extensive cellulitis of the mouth and neck had followed extraction of a septic molar tooth. The cellulitis in this case was complicated by a streptococcic septicemia, and the patient was treated with the calcium salt of penicillin.

Case 1.—A girl, aged 12 years, was admitted to the hospital because of a rather extensive phlegmon of the mouth. One week before her admission she had noticed pain and distress in the region of the left lower first molar tooth. After the onset of pain in the region of the molar tooth, swelling had developed on the left side of the face, mouth, and submaxillary region. Severe pain in the entire left side of the face had been present. On the fourth day of her illness she had been unable to swallow solid foods. The infection had continued to spread and the signs suggestive of general sepsis had developed. At the time of her admission to the hospital she appeared to be seriously ill. Examination revealed redness, tenderness, and marked induration extending from the left side of the face far down into the neck. The mouth could scarcely be opened. It was extremely difficult for her to swallow liquids but the airway seemed adequate (Fig. 1, *a*). The left lower first molar tooth was badly decayed. Her temperature was 101° F. The leucocytes numbered 12,900 per cubic millimeter of blood. A roentgenogram of the mandible did not disclose any abnormality. Blood cultures made at the time of the patient's admission were negative. During the next twenty-four hours after her admission to the hospital, the pa-

tient's condition became progressively worse and the inflammation became more extensive. Initial treatment consisted in the local application of heat. Twenty-four hours after her admission, penicillin therapy was begun. She received 20,000 Oxford units of penicillin sodium in 1 liter of physiologic salt solution twice daily for the next six and a half days. This was administered by the intravenous drip method previously mentioned. Within forty-eight hours after penicillin therapy was started, the patient was able to swallow without difficulty and her general condition was markedly improved. The swelling and edema rapidly subsided. A small abscess formed and ruptured spontaneously through the skin. The temperature reached normal four days after the administration of penicillin was started. The leucocyte count also was normal at this time and remained so. With the exception of a slight amount of drainage, her progress was entirely satisfactory and she was dismissed from the hospital on the fourteenth day after her admission. An abscess was present below the first left molar tooth, which was extracted later by her family dentist. No complications followed (Fig. 1, *b*).

Fig. 1.—Patient in Case 1: *a*, before administration of penicillin was started; *b*, at time of dismissal from the hospital.

Case 2.—The patient was a man, 23 years of age. Two days after extraction of the third, right, mandibular molar tooth, the right side of the patient's face had become painful and extensively swollen. Sulfathiazole and sulfapyridine had been administered in doses that are considered adequate for ordinary therapeutic purposes. When the patient was seen at the Mayo Clinic, the inflammation had extended deeply into the neck and he was critically ill. He was unable to swallow and was experiencing great difficulty in breathing.

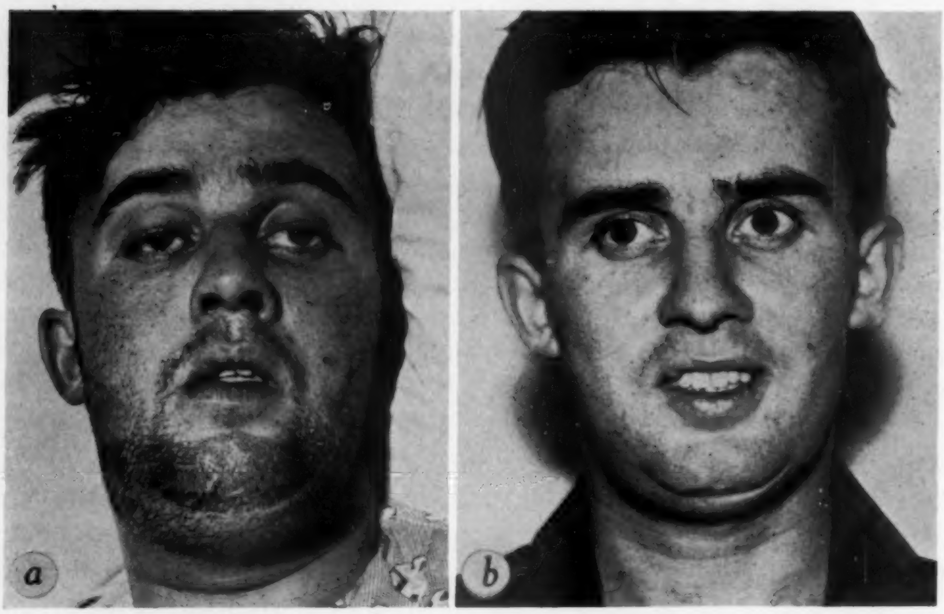

Fig. 2.—Patient in Case 2: *a*, at time of admission to hospital; *b*, at time of dismissal from hospital.

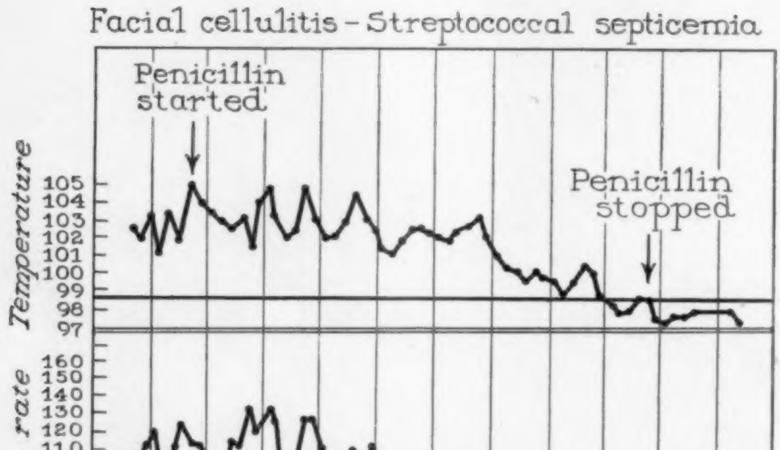

Fig. 3.—Temperature and pulse rate in Case 2. Temperature was taken by rectum for the first seven days; thereafter, it was taken by mouth.

The leucocyte count was 25,000 per cubic millimeter of blood. He could scarcely open the mouth (Fig. 2, *a* and *b*). The rectal temperature was 103° F. but rose gradually to 105° F. (Fig. 3). Blood cultures were taken and the intravenous administration of penicillin calcium was begun (22,000 Oxford units of penicillin calcium in 1 liter of physiologic salt solution were administered twice a day). The blood cultures disclosed the presence of streptococci which produced slight hemolysis in blood agar. Brain broth cultures also showed the presence of streptococci.

During the first twenty-four hours of treatment the patient did not appear to be improving, and on one occasion he nearly died as a result of respiratory embarrassment. Forty-eight hours after initiation of penicillin therapy, there was profuse drainage from the tooth socket and the patient was able to swallow liquids. His temperature and pulse remained elevated. By the fourth day of treatment he was making remarkable progress. A blood culture obtained on this day was negative. He continued to improve. His temperature gradually reached normal after eight days of treatment. By the end of the first week's treatment the patient was able to eat solid foods and felt entirely well. His total stay in hospital was fourteen days.

SUMMARY

Penicillin has been used in six cases of rather extensive cellulitis of the mouth. In two cases the cellulitis was complicated by bacteremia. The use of penicillin did not produce any toxic reaction. The rather extensive cellulitis present in these cases responded almost dramatically to the use of penicillin. If subsequent studies show that penicillin therapy will accomplish satisfactory results in such cases, it may be possible to avoid extensive and radical surgical procedures which have often been necessary. It seems likely that the use of penicillin may shorten the period of convalescence and reduce the hazard of complications associated with cellulitis of the mouth.

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USE OF HIGHER THAN USUAL CONCENTRATIONS OF PROCAINE HYDROCHLORIDE IN DENTISTRY

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PROCAINE hydrochloride is the local anesthetic agent used most frequently in dental operations. In a few dental procedures procaine cannot be used even though such an anesthetic agent is indicated. Practice has tended to dictate the use of 2 per cent concentrations of procaine; the availability and uniformity of solutions free from contamination, offered by packaged preparations, has helped to establish and limit the concentration for dental use.

Austin and Stafne,[1] Lundy,[2] and Stafne[3, 4] have recognized the limitations of 2 per cent solutions of procaine and have given some of the indications for solutions of greater concentration. Trauner[5] reported use of a 4 per cent solution for root resection because sometimes there is pain after use of a 2 per cent solution.

In dental procedures in which procaine is indicated, such as in the extirpation of pulps, the preparation of extremely sensitive gingival cavities, the surgical removal of gingival tissues, in difficult extractions, or in cases in which the teeth to be removed are tender to percussion, have a pulpitis or have periapical granulomas, the failure of 2 per cent solutions to provide anesthesia may be avoided by the use of stronger solutions. Concentrations of procaine greater than 2 per cent were first used by the Section on Dental Surgery at the Mayo Clinic more than ten years ago, owing to the occasional failure of a 2 per cent solution to provide adequate anesthesia. Since then, concentrations of procaine greater than 2 per cent have been employed in many dental operations, especially in cases in which it has been possible to predict the inefficacy of a 2 per cent solution. When failure to obtain profound anesthesia has necessitated a second injection, higher concentrations have been used, since by experience it had been found that the second injection of the same concentration of procaine did not increase the depth of anesthesia. Solutions of 3 or 4 per cent have been most commonly employed, although higher concentrations have been used.

STUDY OF 500 CASES

To obtain data concerning the safety with which higher than usual concentrations of procaine may be used in dentistry, 500 consecutive cases in which injections of higher than 2 per cent solutions of procaine hydrochloride were employed were reviewed. The clinical and laboratory findings were available in each case. The safety with which increased concentrations of procaine can be used can be better evaluated in the light of the clinical and laboratory findings.

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There were 315 females and 185 males, ranging from 13 to 78 years of age, in this series. Two hundred thirty-seven patients were less than 40 years of age, and 263 patients were 40 years of age or more (Table I).

TABLE I

AGE DISTRIBUTION

AGE (YEARS)	CASES
13-19	23
20-29	124
30-39	90
40-49	111
50-59	101
60-69	44
70-79	7
Total	500

TABLE II

EXEMPLARY CASES IN WHICH INJECTION OF THE ANESTHETIC AGENT WAS MADE ON THE OPERATING TABLE: PROCAINE HYDROCHLORIDE ALONE OR PROCAINE HYDROCHLORIDE WITH EPINEPHRINE HYDROCHLORIDE IN A DILUTION OF 1:120,000 WAS USED

SEX	AGE (YEARS)	BLOOD PRESSURE, MM. HG		DIAGNOSIS
		SYSTOLIC	DIASTOLIC	
F	67	260	100	Hypertensive heart disease
F	68	220	100	Hypertension with diffuse arteriolar disease
F	62	240	120	Diffuse arteriolar disease with myocardial and renal degeneration
M	58	220	140	Hypertension, grade 3; peripheral sclerosis, grade 2; possible coronary occlusion about 2 years previously
F	62	240	120	Diffuse arteriolar disease with early renal and myocardial degeneration (hypertension, grade 2)

TABLE III

ONE HUNDRED TWO CASES IN THE SERIES IN WHICH SURGICAL RISK WAS INCREASED

DIAGNOSIS	CASES	DIAGNOSIS	CASES
Hypertension		Diabetes mellitus	
Grade 1	32	Grade 1	6
Grade 2	14	Grade 2	2
Grade 3	6	Grade 3	2
Grade 4	1	Grade 4	4
Addison's disease	1	Nephritis	
Asthmatic bronchitis	1	Acute	3
		Chronic	5
Drug allergy	2	Adenoma of thyroid gland	7
Pregnancy*	2	Myxedema	1
Menopausal symptoms	6	Ménière's disease	1
Latent syphilis	3	Lymphedema	1
Residual poliomyelitis	2		

*Of six and eight months' duration.

The number of patients in the third decade of life is accounted for by the number of third molar impactions for which removal is indicated in this decade. A total of 1,398 teeth was removed in these 500 cases, of which 303 were impacted. Of the teeth removed, 771 were from the maxillary arch and 627 from the mandibular arch. One hundred seventy-two dental roots were

removed, twenty-seven cysts were operated on, and five operations were performed for plastic closures of alveolo-antral fistulas and exploration. Many of the patients had more than one operation.

In this study, a 4 per cent solution of procaine hydrochloride with epinephrine hydrochloride (1:60,000 dilution or less) has been used most often. Occasionally, solutions of procaine hydrochloride exceeding 4 per cent have been used. In some cases the injections were made with the patient lying on the operating table, and procaine hydrochloride was used without epinephrine (Table II). In all cases, the solution, the rate of injection, and regions injected were handled in the manner customary for less concentrated solutions, namely: infiltration anesthesia for all regions of the maxilla, and the inferior dental block plus local infiltration into the immediate site of operation for hemostasis for the mandible. Anesthesia has been profound, and contrary to what might be expected, the duration has not been protracted materially. In no instance has the postoperative course in these cases been influenced by the use of an increased concentration of procaine.

In addition to the cases grouped in Table III, various other cases were of sufficient interest so that I am reviewing them separately.

Case 1.—A woman, aged 49 years, who had a blood pressure of 150/90, and on whom a diagnosis of polycythemia vera had been made, had been operated on previously for carcinoma of the breast, Grade 4 (Broders' grading). Dental operation consisted of removal of seven teeth. The only postoperative complication was hemorrhage, which was anticipated.

Case 2.—A man, aged 27 years, who had Hodgkin's disease, underwent dental surgery satisfactorily.

Case 3.—The patient was a woman, aged 50 years, whose blood pressure was 130/80. A diagnosis of recent coronary thrombosis with anterior infarction had been made. Dental surgery was performed successfully. Procaine hydrochloride was injected after the patient was on the operating table, and a vasoconstrictor agent was not used.

Case 4.—A man, aged 62 years, underwent dental operation. His blood pressure was 130/84. A diagnosis of arteriosclerotic heart disease with indeterminate auricular fibrillation and flutter had been made previously. The patient experienced no untoward results following dental surgery performed under mandibular block anesthesia.

Case 5.—The patient was a man, aged 52 years. The blood pressure was 136/80. A diagnosis of hypertensive and coronary heart disease with congestive failure had been made. Two dental operations were performed, at which a total of thirteen teeth were removed. The postoperative course was uneventful after both operations.

Case 6.—The patient, a woman, aged 64 years, had a blood pressure of 130/80, with arteriosclerosis of the central nervous system. She gave a history which indicated the possibility of a cerebrovascular accident eight weeks previously, and she had the characteristic features of early parkinsonian disease. An uneventful course, however, followed dental surgery.

Case 7.—The condition of a man, 49 years old, whose blood pressure was 218/130, was diagnosed as hypertension, bilateral polycystic kidneys with renal

insufficiency. His blood urea was 116 mg. per 100 c.c. (normal 10-40 mg. per 100 c.c.). Three teeth and one dental root were removed without disturbing the patient.

Six patients who underwent dental surgery had had recent major surgical operations. Although they were convalescent and in various stages of debility, no untoward reactions occurred. The findings for the remaining 385 patients in this study were well within normal limits and were not considered as constituting any unusual surgical risk.

COMMENT

The lowest possible concentrations of procaine hydrochloride have been used in dentistry. Too often the failure to obtain sufficient depth of anesthesia has had a direct bearing on the outcome of the work at hand and has instilled in the patient an undesirable attitude toward future dental care. In view of the amount of procaine used in local anesthesia for surgery on other parts of the body, no concern need be felt for the relatively small amounts employed in dental anesthesia. Psychic disturbances too often are mistaken for sensitivity to procaine, as true procaine idiosyncrasy is rare.

The results obtained by the use of higher than usual concentrations of procaine hydrochloride have been entirely satisfactory, particularly in regard to the adequacy of anesthesia and the freedom from untoward reactions, during injection, during operation, or after operation.

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CYSTS ARISING FROM THE MUCOSA OF THE MAXILLARY SINUS AS SEEN IN THE DENTAL ROENTGENOGRAM

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TWO types of benign cysts may have their origin in the mucosa of the maxillary sinus. They may be designated as being either of a secreting or of a nonsecreting type. This article is concerned with the observation, as seen in the dental roentgenogram, of these cysts that may develop from the mucosa of the maxillary sinus. While these cysts are of little direct interest to the dentist, yet if he makes full use of the dental roentgenogram in his everyday practice, he will want to become more familiar with the variations exhibited by these cysts in the maxillary sinus.

Commonly only a small portion of the maxillary sinus, the lower border, is seen in the dental roentgenogram. In this region it is possible that other structures may be superimposed which cause the outline of the sinus to be indistinct in the roentgenogram. The outline of the normal maxillary sinus, as seen in the dental roentgenogram, appears bilaterally. The size of the maxillary sinus may vary considerably. Generally the sinus extends anteriorly to the first premolar tooth and distally to the region of the second molar. However, the mesial and distal limits may extend farther. The lowest portion of the sinus is usually in the first molar region. In many instances in which there are teeth present, the lower border is near the apices of the teeth. There are extreme cases in which the sinus is so large that it appears as though the roots of the teeth protruded into the sinus cavity.

The lining membrane of the maxillary sinus is a pseudostratified, ciliated, columnar epithelium. While it is thinner than that of the respiratory portion of the nostril, it is histologically essentially like the latter.

Most rhinologists are of the opinion that the secreting type of cyst rarely forms in the maxillary sinus and that the nonsecreting type of cyst is quite commonly encountered. When seen early in their development by radiographic examination, both types of cyst appear radiopaque and it is impossible to distinguish one type from another.

Secreting cysts can be considered as inclusion cysts. They are lined with epithelium and filled with mucus.

Nonsecreting cysts might be considered pseudocysts, as they result from accumulation of tissue fluid in the tissue spaces and do not have a well-formed lining that is made up of connective tissue.

Skillern[1] called attention to the formation of cysts in the maxillary sinus in the form of mucoid or retention cysts. He stated: "The mucoid cyst is characterized by single or multiple, semispherical, yellow or whitish protuberances on the floor of the nasal wall of the antrum, ranging in size from a millet

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seed to a walnut. Occasionally they may grow to such an extent as to completely fill the antral cavity. As the name implies, they are occasioned by obstruction to the glandular outlet, due to some form of inflammation in the immediate neighborhood. No symptoms are occasioned by the presence of these new growths in their original state."

Hajek[2] included cysts of the maxillary antrum under inflammatory tumors of the maxillary antrum. He stated: "In most of the cases of chronic inflammation of the mucous membrane of the maxillary antrum, in which cicatricial changes occur, one or more cysts may be found. Usually they are miliary and scattered. Occasionally individual cysts may be larger than a hazel nut. They develop from cicatricial epithelium."

Hardy[3] reported five cases of cysts of the maxillary sinus. He pointed out that they are not encountered frequently and at times are discovered only in the course of a routine radiologic study of the paranasal sinuses. In the five cases reported by Hardy, the cysts were considered as simple cysts of the maxillary sinus. Clinically and pathologically these cysts were not of dental origin. All of the cysts were lined with columnar or cuboidal epithelium.

Fig. 1.—Cysts in the maxillary sinuses of two different patients that have been edentulous for more than ten years. This is evidence that these cysts are not of dental origin.

Lindsay[4] presented a brief classification in which, under the heading of benign cysts arising from the mucosa of the sinus, he distinguished secreting cysts, including gland cysts and mucocele, from nonsecreting cysts. He noted that these cysts appear opaque in the roentgenogram.

He further mentioned that the secreting cyst, commonly described as a mucocele, may develop to huge proportions, invading the orbit and producing extensive deformities of the face. It is found frequently in the ethmoid or the frontal sinus, but rarely in the maxillary antrum.

In his paper he was concerned primarily with the nonsecreting cysts of the mucosa of the maxillary sinus. He mentioned that their occurrence is rather common. They vary in size. They do not cause the deformity with growth that may occur with the secreting type of cyst. They ordinarily rupture spontaneously usually before they reach any large size.

Under the heading of cystic polypoid formations, Finck[5] described mucocele formation and mesothelial cysts, which may be considered comparable to secreting and nonsecreting cysts of the mucosa of the sinus respectively. He described these two cystic polypoid formations as appearing opaque on radiographic examination.

McGregor,[6] dealing with the formation and histologic structure of cysts of the maxillary sinus, pointed out that secreting cysts are caused primarily by

damage to the cilia of the gland tubules by infection, although edema, infiltration and fibrosis may be contributing factors. This type of cyst is lined with epithelium and filled with mucus. It, too, appears opaque on the roentgenogram.

Fig. 2.—Cyst which has been removed from maxillary sinus. The circumscribed and definitely rounded form of the cyst conforms to the characteristic dome-shaped radiopaque image that it produces in the dental roentgenogram.

Fig. 3.—Cysts in the maxillary sinus. Whether these cysts are large or small their characteristic radiopaque and dome-shaped outline is always quite definite when seen in the dental roentgenogram.

Fig. 4.

Fig. 4.—A cyst of considerable size in the maxillary sinus. On the left, when first noted. On the right, when seen sixteen months later. The cyst has increased in size to a minor degree.

McGregor, too, preferred to regard the nonsecreting cyst as a mesothelial cyst as referred to by Finck. He stated that these types of cysts "occur in edematous mucous membrane and are due to the accumulation of tissue fluids in the tissue spaces. They have not a truly formed wall. The edema is due to a disturbance in the water balancing mechanism, caused by infection." They are filled with tissue fluids. Also, they appear opaque on the roentgenogram.

It is possible for either of these types of cysts to occur any place in the maxillary sinus. However, they would be seen in the dental roentgenogram

only when they develop from the mucosa in the floor of the maxillary sinus and only if the sinus outline appears clear and distinct in the dental roentgenogram. For, as previously mentioned, customarily only a small portion of the maxillary sinus, the lower border, is seen in the dental roentgenogram. Because these cysts do occur at any place within the maxillary sinus, it is quite evident that, from both a clinical and a pathologic standpoint, they are not of dental origin. This is further emphasized by those cases in which the patient is edentulous and in which cysts are observed in the sinus as in Fig. 1.

When seen, these cysts are readily recognized, especially if there are no superimposed bony structures and the sinus appears clear or radiolucent on the roentgenogram (Figs. 2, 3, and 4). They appear radiopaque against a sinus background that is radiolucent. Their outline form as seen in the roentgenogram has been described aptly as dome-shaped. In the dental roentgenogram they are seen arising from a base, the floor of the maxillary sinus, to assume a half-moon or half-round shape.

The presence of one of these cysts has been observed in the maxillary sinus of one patient for a period of six years. During that time there has been no apparent change of size. Yet other cysts have been observed that sometimes disappeared during the interval of a year.

It is possible for one of these cysts to grow to such a size that the limits of the cyst outline cannot be seen in the dental roentgenogram. In such instances the presence of the cyst would not be recognized, except that it might give a cloudy appearance to the antrum in the dental roentgenogram.

In the examination of 600 consecutive patients who had complete dental roentgenograms, twenty-four of these patients had a cyst in the maxillary sinus as determined by dental radiographic findings.

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CYSTS OF THE JAWS LINED WITH CILIATED COLUMNAR EPITHELIUM

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A RADICULAR cyst usually is lined with stratified squamous epithelium which is derived from the epithelial rests of Malassez in the periodontal membrane. The lining of a follicular cyst is derived from the enamel epithelium of the tooth germ.

Median anterior maxillary cysts are of developmental origin. The lining of the cyst varies with the site of origin along the epithelial cord of the incisive canal. Stafne, Austin, and Gardner[1] have shown that cysts originating from the superior portion of the epithelial cord are lined with ciliated columnar epithelium; cysts originating from the midportion are lined with a transitional type of epithelium, and squamous epithelium is found to line cysts which develop from the part of the cord arising from the oral cavity. These cysts may give rise to daughter cysts (Fig. 1). Another developmental cyst, sometimes found in the mouth, is the dermoid cyst, which is lined with epidermis.

Traumatic, or hemorrhagic, cysts occur within the medullary portion of the jaw and usually have no true lining, although fibrous tissue may be found in parts of the bone cavity. Cahn[2] stated that all cysts of the jaw bones arise from embryonal epithelial remnants, and he described sequestration dermoid cysts.

The occurrence, in the jaws, of cysts, which are lined with ciliated columnar epithelium, other than median anterior maxillary cysts, is rare. Bland-Sutton[3] reported that columnar cells are found especially in cysts that are associated with the first permanent molar. The presence of both squamous and ciliated columnar epithelium in a granuloma on a tooth the identity of which had been lost has been reported by McConnell.[4] He suggested that the tooth probably was from the maxillary arch, and that the maxillary sinus may have been perforated. Adloff (quoted by McConnell) found ciliated columnar epithelium in two cysts of the maxilla. In thirty-five of fifty-four cases of pulpless teeth, Stein[5] found epithelium in the periapical region. Ciliated columnar epithelium was present in some cysts, and in each case a fistula communicated with the maxillary sinus.

The maxillary sinuses play an important part in the origin of noncongenital cysts that are lined with ciliated columnar epithelium and, indeed, might themselves be mistaken for cysts. According to Thacker-Neville,[6] the maxillary sinuses may be absent, small, or subdivided into two. Brophy,[7] who quoted Gruber, said that a thin lamina of bone is found crossing the maxillary sinus in 2.5 per cent of the cases. This percentage agrees with that mentioned by Schaeffer[8] if the posterior ethmoidal cells are also counted with the maxillary sinuses. The doubling of the maxillary sinus is explained in Schaeffer's observa-

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tion of two pouches (anlage of the sinus) growing side by side from the ethmoidal infundibulum. Such an initial doubling explains some of the duplications of the ostium maxillare.

Cysts may arise within the maxillary sinus. Brophy pointed out that an occlusion of the normal opening through the antronasal wall (the ostium maxillare) may cause a retention of mucus, and the filling of the sinus may in turn be followed by absorption of its bony walls and the development of a typical cyst, a so-called hydrops of the sinus. In describing the formation and histologic structure of cysts of the maxillary sinus, McGregor[9] discussed: (1) the role of infection, which causes damage to the cilia of the tubules of the glands, as a primary cause of secreting cysts, with edema, infiltration, and fibrosis acting as possible contributing factors; (2) dental cysts that arise from epithelial rests of Malassez, which become stimulated into activity by infection, and (3) mesothelial cysts, which occur in an edematous mucus membrane owing to the accumulation of tissue fluid in the tissue spaces. These mesothelial cysts do not have a truly formed wall.

Fig. 1.—Daughter cyst arising from a median anterior maxillary cyst.

Kronfeld[10] has given a clear and definite explanation of the presence of ciliated columnar epithelium in cysts of the jaw. Since this type of epithelium is not normally encountered in the jaw, one can conclude that it came from without the jaw. Proliferation of ciliated columnar epithelium of the maxillary sinus into apical abscess cavities, or radicular cysts which communicate with the maxillary sinus, accounts for the finding of this epithelium in cysts of dental origin.

Examination of dental roentgenograms does not always clearly reveal the presence of a dental cyst in the region of the maxillary sinus. Variation in the distance between the floor of the sinus and the apices of the teeth, the presence of incomplete or complete septums in the sinus, the pneumatization of the sinus as seen in both dentulous and edentulous jaws are but a few of the examples of the factors that make a positive diagnosis of cysts in the maxilla difficult.

More than 600 sections were examined microscopically in an attempt to find cysts of the jaws that were lined with ciliated columnar epithelium. These sections were obtained in 389 cases in which operations were performed on the jaws. A cyst that was lined with this type of epithelium was found in only one case (Case 1).

REPORT OF CASES

CASE 1.—A woman, aged 46 years, came to the Mayo Clinic eighteen years ago complaining primarily of pain over the right eye and right cheek, which had begun eight months previously. Examination failed to reveal any reason for

Fig. 2.—Dental roentgenograms showing the maxillary arch with cuspids unerupted.

the pain. Puncture of the right maxillary sinus did not disclose any abnormality. Roentgenographic examination revealed a circumscribed shadow over the right maxillary sinus and the possibility of a polyp was considered. The patient was advised to have all of her teeth removed. The dental roentgenograms showed an

encysted shadow at about the line of the apices of the teeth, but very definitely below the floor of the maxillary sinus. Investigation was recommended by the Section on Otolaryngology and Rhinology. A cyst was removed from the right maxillary bone. A section was sent to the pathologist for microscopic examination. A report of a simple cyst associated with marked inflammation was returned. In studying sections of this cyst, I found that the lining of the cyst

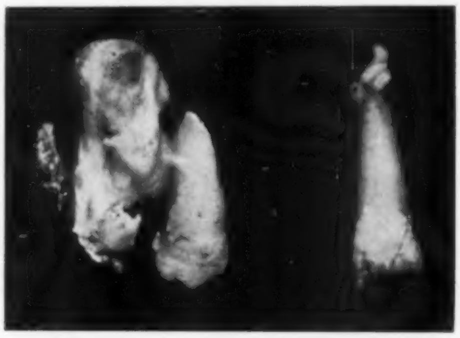

Fig. 3.—Left cuspid with granuloma. Right cuspid showing point of contact with the cyst.

Fig. 4.—Section through granuloma at apex of left cuspid (Fig. 3).

consisted chiefly of ciliated columnar epithelium. The dental roentgenograms were not available. There is no record of a possible communication with the maxillary sinus.

CASE 2.—A man, aged 44 years, had had recurrent swelling and occasional pain, similar to a toothache, in the region of the left maxillary cuspid. Clinical

examination revealed an edentulous maxillary arch with some inflammation at the crest of the ridge in the left cuspid position. Dental roentgenograms showed that both maxillary cuspids were present and unerupted (Fig. 2). The cuspid on the left showed some changes of the crown and a periapical rarefaction. Removal of this tooth had been attempted six years previously. The right cuspid showed little, if any, change. A cyst extended from the right cuspid distally about 3 cm. and had displaced the maxillary sinus. At the lower border of the cyst and near the crest of the alveolar ridge, a root fragment was present. The possibility of this being a radicular or possibly a dentigerous cyst was considered. The unerupted cuspids and the dental root were removed. The cyst associated with the right cuspid extended posteriorly and occupied at least two-thirds of the maxillary sinus. No communication between the cyst and the sinus was demonstrable. Specimens were sent for microscopic study.

Fig. 5.—Ground section of the crown of the left cuspid photographed (by reflected light).

When the left cuspid was removed, a granuloma was found (Figs. 3 and 4). As this tooth was prepared, pulpal degeneration was evidenced by the foul odor which attended sectioning. The ground section shows the carious process which probably developed as a result of the attempted removal six years previously (Fig. 5). The pathologist reported that the tissue around the crown consisted of a cyst that was lined with epithelium and was the site of severe inflammation.

Upon examination following the removal of the right cuspid and the cyst, the point of attachment was found to be at about the mid-point of the root portion of the tooth and not at the cemento-enamel junction as would be the case in a follicular cyst (Fig. 3). The crown and root tip were removed, and the remaining portion of tooth with the attached cyst was decalcified. The progress of decalcification was checked roentgenographically according to a method

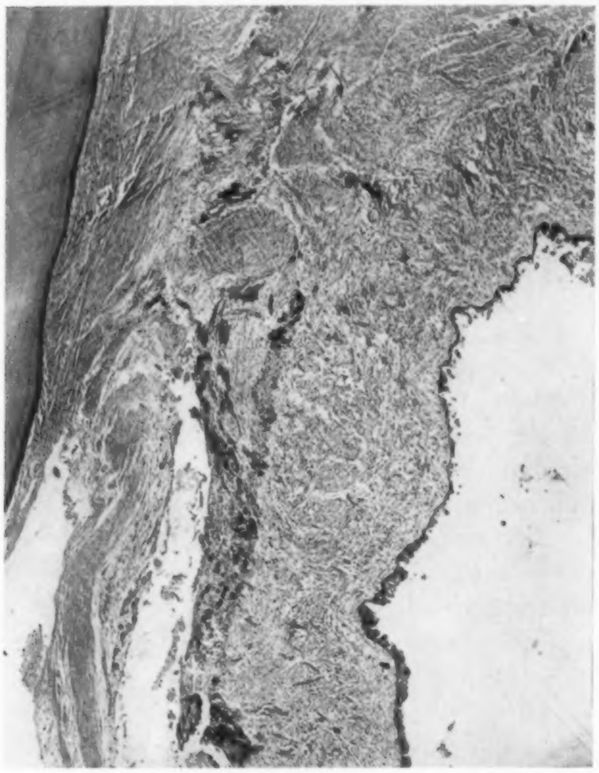

Fig. 6.—Section of cyst and right cuspid. The thick fibrous wall of the cyst is in no way related to the tooth.

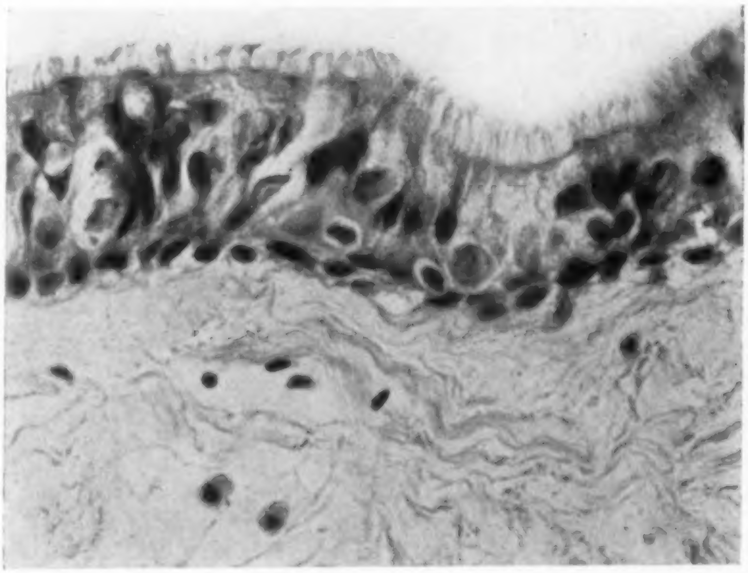

Fig. 7.—Higher magnification of cyst wall (Fig. 6) showing the ciliated columnar epithelium in detail.

previously reported.[11] Sections made from this specimen showed that the cyst existed independently of the tooth (Fig. 6) and that the lining consisted chiefly of ciliated columnar epithelium (Fig. 7).

COMMENT

Few unerupted teeth ever give rise to a true odontalgia unless at some previous time an opening into the oral cavity has existed, as it did in Case 2. In this case, caries resulted in pulpal degeneration and the formation of a periapical granuloma on an unerupted tooth. The cyst in the right maxilla proved interesting because of its size, position, and association with both a tooth root and an unerupted tooth. The presence of ciliated columnar epithelium suggests an early communication with the maxillary sinus. The possibility that the cyst in Case 2 was a median anterior maxillary cyst is ruled out by the distance of the cuspid from the midline of the maxilla and the occurrence of the cyst distal to the cuspid tooth; without the existence of a true median anterior maxillary cyst, this cyst could not be a daughter cyst. Probably the root fragment is the etiologic factor in this case. Were it not for the thickness of the lining of the cyst and the presence of a dental root, one might be tempted to suggest that this could be an antrum separated by a complete bony septum from the sinus situated posteriorly. One other possibility that must be kept in mind is that such a cyst might be the maxillary sinus proper, and the sinus situated distally could be a large posterior ethmoidal cell.

Were routine sections to be made of periapical tissues and examined for the presence of epithelium resembling that of the maxillary sinus, more instances of these cysts would be found. In this study of 389 cases, no instances have been found in which cysts of the mandible have been lined with ciliated columnar epithelium. No basis for such an occurrence exists. Other than the branchial cleft cysts in the region of the angle of the mandible, there is no reason to expect to find a cyst of the mandible with a ciliated columnar lining.

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CYSTIC ODONTOMA

Report of Case

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THE following case, in which a dentigerous cyst has its origin in an odontoma, is of interest because the roentgenographic evidence was not particularly suggestive of this type of cyst.

REPORT OF CASE

A woman, 42 years of age, was seen in a section on general medicine at the Mayo Clinic. She had had intermittent pain and swelling in the anterior region of the left maxilla and at times it had been difficult for her to wear a denture. She first had noted the swelling about six months previously, and she had become concerned because she had felt that the swelling gradually had become larger. She was then referred to the section on Dental Surgery.

The upper jaw had been edentulous for fifteen years. Clinical examination revealed a definite bulging on the labial surface of the maxilla. It was most prominent over the lateral incisor region and extended upward and above the labial fold. The overlying mucous membrane was inflamed, no doubt owing to the increased pressure of the artificial denture on the swollen surface.

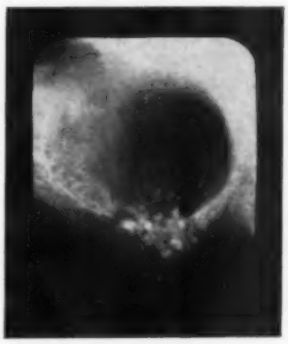

Fig. 1.—Roentgenogram of cyst showing radiopaque bodies which proved to be an odontoma.

Roentgenographic examination revealed a cyst which was about 3 cm. in diameter and extended from the midline posteriorly to the anterior wall of the maxillary sinus. At the lower border of the cyst several small radiopaque bodies were seen (Fig. 1).

The cyst was removed with local anesthesia. An incision was made on the labial surface. Retraction of the mucoperiosteum revealed a thin labial bony plate overlying the cyst, which at some points was on the verge of being perforated. Sufficient bone was removed to allow complete enucleation of the cyst. At the lower border of the cyst was a mass which felt hard and gritty.

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Microscopic examination revealed that the wall of the cyst was composed of a connective tissue of average thickness, and that it was lined with squamous epithelium. Parts of the wall were inflamed. One portion of the wall, however, was markedly thickened, and within this part of the wall numerous calcified dental structures were seen (Fig. 2). The diagnosis was cystic odontoma.

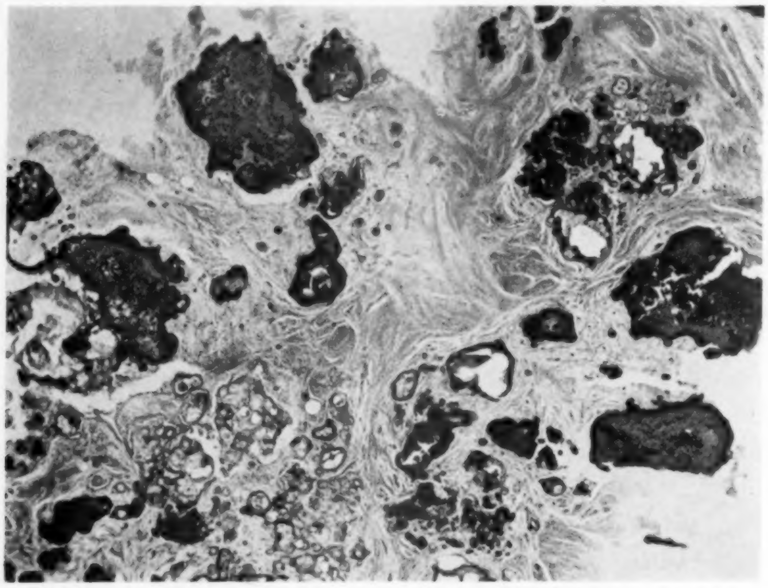

Fig. 2.—Dental structures within the wall of the cyst (×23).

COMMENT

The most likely explanation for the origin of this cyst is that it developed from enamel epithelium present in the odontoma. In this respect it is similar to dentigerous cysts that develop from the enamel epithelium of unerupted teeth. It is doubtful if a diagnosis of a dentigerous cyst could have been made from the roentgenogram, for the scattered radiopaque bodies more closely resembled foreign bodies than an odontoma.

A CHARACTERISTIC DENTAL FINDING IN ACROSCLEROSIS AND DIFFUSE SCLERODERMA

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THIS report deals with a dental roentgenographic finding which to our knowledge is peculiar only to patients suffering from acrosclerosis and diffuse scleroderma. This roentgenographic finding is an increase in the width of the periodontal space which, when it does occur, is sufficiently characteristic to cause the presence of these conditions to come to mind immediately. In a large measure this statement can be substantiated by the fact that since this characteristic feature was first observed several years ago, a tentative diagnosis of acrosclerosis or scleroderma has been made from the dental roentgenogram in four cases at the Mayo Clinic. In these four cases the tentative diagnosis was confirmed later from the history and the record of the general physical examination.

From a review of dental roentgenograms made in 127 cases of acrosclerosis or diffuse scleroderma, the characteristic widening of the periodontal space was found in nine (7.08 per cent) of the cases. Four of the patients were males and five were females; the average age was 36.9 years. In eight of these cases a diagnosis of acrosclerosis had been made, and in the remaining one the diagnosis was diffuse scleroderma. In the case of diffuse scleroderma, the dental feature was less marked than in the eight cases of acrosclerosis.

In diffuse scleroderma the entire surface of the skin may be involved. In acrosclerosis the disease is chiefly of the fingers, face, shoulder girdle, and upper part of the thorax, and sometimes, too, sclerosis of the mucous membranes may be present.[1] Acrosclerosis usually is associated with Raynaud's disease. When the face has become involved, the patient is unable to open the mouth as wide as formerly. In this disease too there may be roentgenologic evidence of resorption of bone, particularly of the distal phalanges, which may progress to leave a shortened phalangeal stump. Occasionally fragments of bone may become separated from the distal phalangeal tips.[2] Prinzmetal[3] is of the opinion that this osseous resorption is caused by continuous pressure of the involved skin.

The amount of increase varies in the width of the periodontal space in the dental roentgenogram, which is the characteristic dental feature in acrosclerosis and scleroderma (Fig. 1). All of the teeth of a person may not be affected and the posterior teeth are involved more often than the anterior teeth. On the involved teeth, the space which has been created surrounds the entire root of the tooth and is almost uniform in width. The increase in width of the periodontal space of involved teeth produced by acute pericemental inflammation, pyorrhea alveolaris, and traumatic occlusion is not so strikingly uniform, and this lack of uniformity in width permits a distinction of these conditions in the

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dental roentgenogram from acrosclerosis and scleroderma. The roentgenographic appearance of the teeth in acrosclerosis and diffuse scleroderma is somewhat similar to that of teeth which are rapidly extruding from their alveolar sockets. On clinical examination, however, it will be found that apparently the involved teeth are not extruded, for on closure these teeth are on the same plane of occlusion as the teeth which are not involved. The increase in size of the periodontal spaces obviously is created at the expense of the alveolar sockets, for there is no roentgenographic evidence of resorption of the roots, and the roentgenogram reveals marked changes in the wall of the socket. The normal lamina dura is obliterated. In some instances there is roentgenographic evidence of active resorption with complete destruction of the normal wall. In others a wide radiopaque line outlines the limits of the enlarged socket, which suggests that resorption has been followed by the formation of a dense and sclerotic wall for the alveolar socket which is appreciably wider than a normal wall of the socket.

Fig. 1.—Typical widening of the periodontal space in dental roentgenograms selected from four patients suffering from acrosclerosis.

On clinical examination in the nine cases which form the basis of this report, the teeth were surprisingly firm in their sockets. In view of the extent of increase in size of the alveolar socket, a great deal more motility of the teeth would be expected ordinarily. With but few exceptions, and only when periodontosis was associated, was the gingival attachment broken. In most instances the gingival crevice was of normal depth, and the gingival attachment to the root of the tooth was unbroken. At the time of dental examination we failed to note carefully whether there was undue sclerosis of the overlying mucous membrane in the nine cases in which dental involvement was present. The ability of some patients to open the mouth only partially made it difficult to obtain a good position for the intraoral dental films when the roentgenograms were made.

PATHOLOGIC CHANGES

Tissue available for microscopic study consisted of an upper left second premolar and a lower left second premolar tooth that were extracted in one of the nine cases. This patient, a woman, 39 years of age, presented the characteristic cutaneous signs of acrosclerosis. When the teeth were delivered, it was found that the roots were covered by an excessive amount of soft tissue which was firmly attached to them. This abnormally thick tissue was noted immediately, because that part of the periodontal membrane which is normally attached to the surface of the root of an extracted tooth is extremely thin and can be observed on the surface of the extracted root only on close inspection. A roentgenogram of the upper left second premolar tooth is shown in Fig. 2, *a*. A longitudinal section through the center of the tooth is shown in Fig. 2, *b* to illustrate the approximate thickness of the membrane attached to the root. At some points the membrane was more than 1 mm. thick; it was probably thicker than shown in Fig. 2 for a portion of it no doubt remained attached to the wall of the alveolar socket.

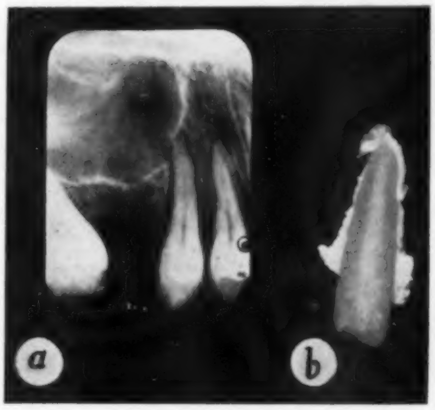

Fig. 2.—Upper left second premolar tooth in a case of acrosclerosis: *a*, roentgenologic appearance; *b*, section of extracted premolar showing attached periodontal membrane.

After removal, the upper left second premolar tooth was decalcified and longitudinal sections were prepared for microscopic study. In some sections the gingiva was present. Neither gingivitis nor pyorrhea alveolaris was present, for the epithelium in the gingival crevice was intact, and there were no inflammatory cells in the subepithelial connective tissue. The sections shown in Figs. 3 and 4 are from the coronal portion of the root and near the bottom of the gingival crevice. They illustrate the abnormally thickened periodontal membrane. The collagenous fibers of the periodontal membrane were firmly attached to the cementum and extended diagonally away from its surface. Ordinarily, the fiber bundles are continuous from the cementum to the alveolar bone, but in this condition the continuity of the fibers is broken at some distance from the cementum. In the coronal portion shown in Figs. 3 and 4, however, the fibers of the periodontal membrane conformed most closely to a normal arrangement. In the middle third the direction of most of the fibers was parallel to the long axis of the root, and in the apical region there was little semblance to the normal arrangement of fibers. The cementum was of uniform thickness

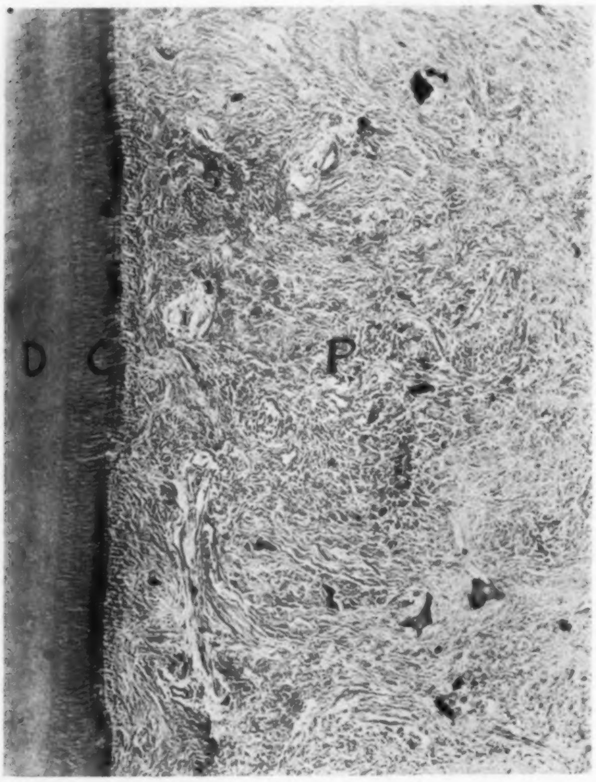

Fig. 3.—Markedly thickened periodontal membrane; *D*, dentine; *C*, cementum; and *P*, periodontal membrane (×60).

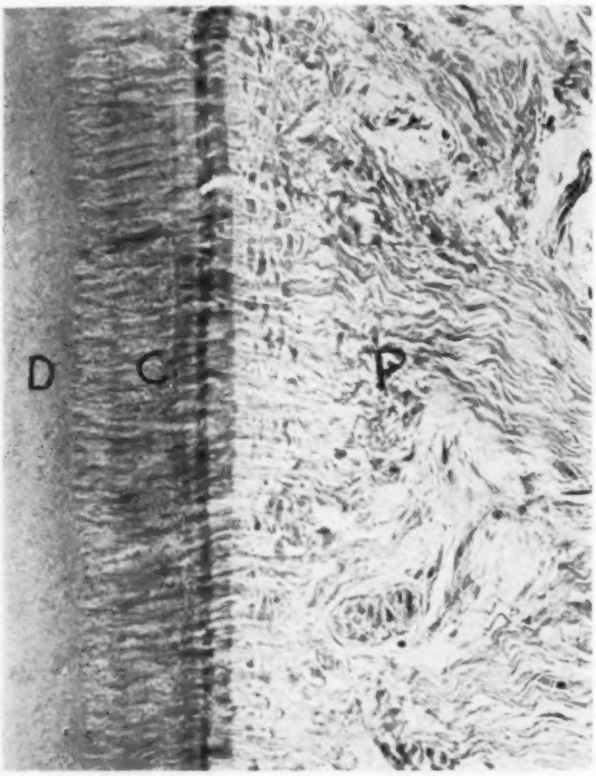

Fig. 4.—Higher magnification of region shown in Fig. 3: *D*, dentine; *C*, cementum; and *P*, periodontal membrane (×235).

around the entire root and was of the primary type. No secondary cementum was present nor was there any evidence of resorption of the cementum. The walls of the blood vessels in the periodontal membrane were thickened,[4] a change which is also one of the histologic characteristics of the vessels in the skin in scleroderma and acrosclerosis.

Comment.—According to Kronfeld,[5] the average thickness of the periodontal membrane of a normally functioning tooth is from 0.18 to 0.25 mm. Observations on the influence of function on the thickness of the periodontal membrane also have been made, and it has been found that the less the function, the thinner the periodontal membrane. In some instances, the average thickness of the periodontal membrane of a nonfunctioning tooth is only half that of the membrane of a tooth that is in normal function.[6] Obviously, in scleroderma when the face is involved and motion of the jaw is limited, function also must be reduced. The logical assumption, therefore, is that the periodontal membrane would be thinner than usual in this condition, but the reverse is true. In some instances, the width of the membrane actually is increased from two to four times that of the periodontal membrane of a tooth in normal function.

SUMMARY

A characteristic change has been observed in the dental roentgenograms of patients suffering from acrosclerosis and diffuse scleroderma, in which the prominent feature is a widening of the periodontal space. This widening of the space apparently occurs most often in association with acrosclerosis although it also may be present with diffuse scleroderma.

Microscopic examination of one specimen revealed that the enlarged periodontal space is occupied by a markedly thickened periodontal membrane. Thickening of the vessel walls suggests that the change in the periodontal membrane is probably brought about by the same circulatory disturbance which is a salient feature in acrosclerosis and diffuse scleroderma.

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DENTAL FINDINGS IN FOUR CASES OF CLEIDOCRANIAL DYSOSTOSIS

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The syndrome cleidocranial dysostosis is of interest to the dental profession because of the unusual dental abnormalities it presents. It is most likely to be observed by the orthodontist or the oral surgeon. Although numerous cases have been reported in the literature, the condition is relatively rare.

Fitzwilliams mentioned that Morand had described the symptoms of this disease in 1766. It was not until a later date, however, that the disease was named by Pierre Marie and Paul Sainton.[1] "On the 14th of May, 1894, Pierre Marie and Paul Sainton presented to the Medical Societies of Hospitals two patients, father and son, both showing similar anomalies: namely, the fontanelles had failed to close; the face appeared to recess on the skull; the superior maxilla was also retracted on the lower jaw; the teeth were of poor shape and irregularly placed; the palatine arch was curved and only partially fused in its median line; the clavicles were reduced to a sternal and an acromial portion, the mid-part being absent.

"One year later Dr. Bolognesi presented to the same group two other patients, mother and daughter, presenting similar anomalies to those described by Marie and Sainton. Because of the similarities, Marie and Sainton concluded this new disease deserved or necessitated a special name. They proposed hereditary cleido-crannienne dysostosis, which term describes succinctly in four words the basic characteristics of the disease."

Although it is generally believed that the anomaly chiefly involves those bones that are formed from membrane, its exact cause is unknown. The literature contains reports of few cases in which only membranous bones were involved. There are usually other bony defects which involve bones derived from cartilage. It is possible, but not probable, that when this does occur it is accidental.

Many of the articles on this subject make reference to the manner in which the maxilla and mandible may be involved and the relation of involvement of these bones to the various disturbances of dentition. A few of the authors who have given attention to the dental findings are Rhinehart,[2] Fitzwilliams,[3] Cooper,[4] Heupel,[5] Rushton,[6] Beeks,[7] Payne,[8] Gulas,[9] and Kostečka.[10]

According to their method of ossification, the bones of the skull may be divided into three types: (1) cartilaginous bones, (2) membrane bones, and (3) cartilaginous bones that develop from the cartilages of the branchial arches. As further described by McMurrich,[11] "The bones belonging to each of these categories are primarily quite distinct from one another and from those of the

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other groups, but in the human skull a very considerable amount of fusion of the primary bones takes place, and elements belonging to two or even to all three categories may unite to form a single bone of the adult skull."

Only the maxillary and mandibular bones and the parts they give rise to will be considered in this study. McMurrich considered the maxilla strictly as a membranous bone; however, he felt that it should be considered primarily as belonging to the branchial arch skeleton. He further cited that both the maxilla and mandible are derived from the first branchial arch. In the maxillary process, no cartilaginous structure is formed, but a membranous bone, the maxilla, is developed in it. Its cartilaginous representative is suppressed by a condensation of the development. The mandibular process is developed from a cartilaginous bar of the first branchial arch, known as Meckel's cartilage. The original cartilaginous bone becomes ensheathed by membrane bone and eventually disappears completely; therefore, one finds that the mandible is essentially a membranous bone replacing a cartilaginous precursor.

This investigation is concerned with the dental findings in four cases as well as with the abnormalities that were found to affect the maxillary and mandibular bones.

REPORT OF CASES

CASE 1.—A white man, aged 43 years, was seen in the Section on Dental Surgery of the Mayo Clinic in the course of a general physical examination. He stated that he had been wearing upper and lower artificial teeth for many years. This proved to be of interest later when dental roentgenograms were obtained of his supposedly edentulous upper and lower jaws. Clinically, it was apparent that while all of the teeth had been removed, several roots remained in the jaws, and that an unerupted tooth possibly was present in the upper right cuspid region. The palate of the upper maxilla was within normal limits as far as height was concerned. Prognathism of the lower jaw, as is often seen in such cases, was not evident in this case. No hereditary history was available.

Roentgenographic examination disclosed unerupted and supernumerary teeth in the upper anterior arch (Fig. 1). The teeth situated in the upper left canine and upper right canine regions were geminated teeth. An unerupted third molar and unerupted premolars were present on the left side of the mandible. This was of interest when it was recalled that this patient had worn artificial dentures for many years. The derangement of these unerupted teeth, as far as position was concerned, was prominent. It also was found that roots still remained in the upper left first premolar, the lower left central incisor, the lower right first premolar, and the lower right second molar regions.

The results of general physical examination was objectively negative except for certain developmental defects. The roentgenograms of the head and cervical segment of the vertebral column revealed bony defects which are pathognomonic of cleidocranial dysostosis. Other laboratory studies did not disclose anything of significance.

CASE 2.—The patient was a white woman, aged 53 years. She was wearing upper and lower artificial dentures. She had had a great deal of trouble with erupting teeth after all of her teeth apparently had been removed. Her teeth

had been out for a number of years, and, in the interim, she had had four different upper and lower dentures made. Clinically, a small draining fistula was noted in the upper right second molar region. The alveolar ridge was quite broad and the palate was rather shallow. In the lower jaw, the mucous membrane over the alveolar ridge in the left second premolar region was quite in-

Fig. 1.—Unerupted and supernumerary teeth in upper anterior arch (Case 1). Geminated teeth are visible in the upper left canine region and upper right canine region.

flamed. A portion of a crown of a tooth was visible in the right canine, second premolar, and second molar regions. The lower jaw was prognathous.

Roentgenographic examination revealed that a number of unerupted and supernumerary teeth were present in both the upper and lower jaws (Fig. 2).

Many of the teeth were arranged in a most irregular pattern; some were inverted. There was a geminated tooth in the lower right second premolar region.

The patient remembered that a great-aunt of her mother's had been affected in this manner. A daughter of the patient one of two children, also had been affected similarly. Both the patient and her daughter were mentally normal.

Fig. 2.—Unerupted and supernumerary teeth in upper and lower jaws (Case 2). A geminated tooth is visible in the lower right second premolar region.

Because of the irritation and discomfort that occurred when the patient was wearing her artificial dentures, the root fragment was removed from the upper right second molar region. A lower right premolar was removed by simple extraction.

The geminated tooth (Fig. 3) in the lower right jaw was removed also. It was necessary to retract the mucoperiosteum and free the teeth by removal of a considerable amount of the buccal process before this tooth could be removed.

Except for certain developmental defects, the results of general physical examination were negative. Roentgenograms of the head and cervical segment of the spinal column revealed certain osseous defects indicative of cleidocranial dysostosis. Laboratory studies did not disclose any abnormality.

Fig. 3.—Geminated tooth removed from lower right second premolar region in Case 2.

Case 3.—A white girl, aged 15 years, was seen in the Section on Dental Surgery in the course of a general physical examination. Retarded growth and failure of teeth to erupt were the chief symptoms. There was no familial history of any similar disease. A diagnosis of cleidocranial dystrophy had been made before she came to the clinic.

At birth, it was thought that the osseous development of the cranial bones was deficient; however, it was not until the girl was ten years of age that a series of roentgenograms had been made because of retarded growth. Although the long bones had not been involved, osseous defects of the skull and clavicle, cartilaginous changes in the lumbar vertebrae, and involvement of the terminal phalanges of the thumbs and toes had been noted.

Her deciduous teeth apparently had erupted normally. However, a short time before she was ten years of age, failure of the permanent teeth to erupt had been noted (Fig. 4), and orthodontic treatment had been started.

When she had been about 13 years of age, it had been recognized that the orthodontic measures that had been taken three years previously had proved unsuccessful (Fig. 5). The deciduous teeth, therefore, had been removed, but the permanent teeth had not been disturbed.

As the permanent teeth still failed to erupt, upper and lower artificial dentures were placed over the alveolar ridges (Fig. 6).

When seen at the clinic, the patient was wearing an upper and lower artificial denture. The alveolar ridges were quite broad. The palate was closed but the vault was quite high. A small portion of an upper right premolar crown was visible. In the lower jaw on the right side, a portion of the crowns of a premolar tooth and of a molar tooth were noted. It was evident that there was a prognathism of the lower jaw.

Dental roentgenograms, made when the patient was 10, 12, and 15 years of age, were compared. In this five-year period, the failure of most of the perma-

nent teeth to erupt is the salient feature. In the first dental roentgenogram that was made, it would appear that the first molars, as well as some of the other teeth, might erupt normally. However, the dental roentgenograms later revealed that they failed to do so, as there was a tendency for the teeth to tip and incline to one side. Several of the unerupted permanent teeth that were in an

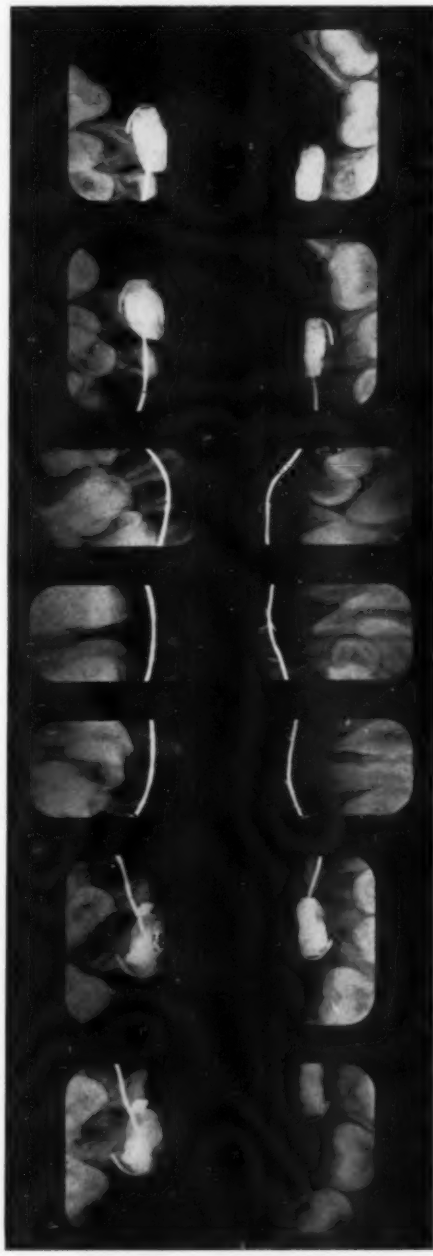

Fig. 4.—Unerupted and supernumerary teeth in Case 3. Roentgenograms made shortly after orthodontic treatment had been started. Patient was about 10 years of age when these roentgenograms were made.

upright position at the age of about 10 years were practically lying in a horizontal position within the body of the jaw when the patient was 15 years of age. The premolars and lower incisors seemingly possessed the greatest eruptive tendency. A lower premolar and an incisor had been removed. The presence of supernumerary teeth was quite evident.

At the age of 10 years and also the age of 15 years, the laboratory findings were normal. Laboratory tests did not disclose any abnormality.

The patient was one of three children. She was the only one affected. The patient's father and mother were both free from the affection. The patient was mentally normal and rated high scholastically, and had a very pleasant personality.

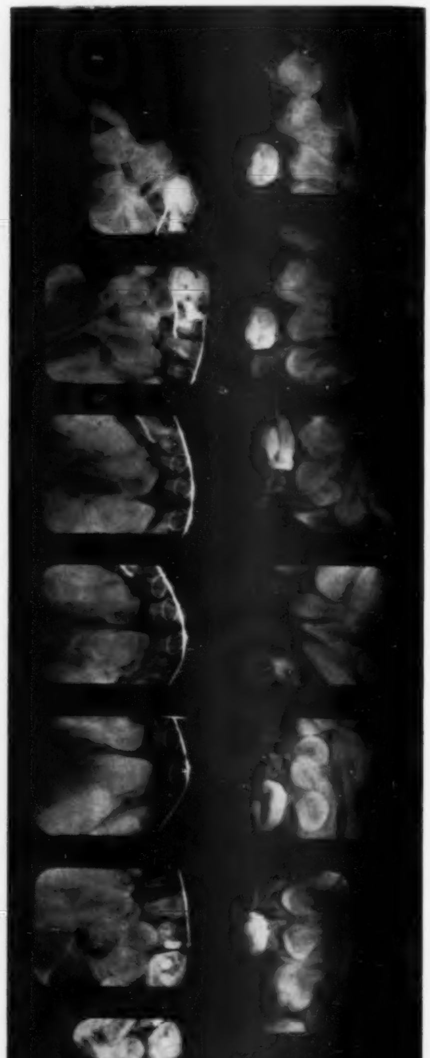

Fig. 5.—Roentgenograms made when the patient in Case 3 was 13 years of age. Resorption of deciduous upper and lower anterior teeth has progressed slowly. The unerupted permanent teeth have become more irregularly situated in the jaws.

CASE 4.—A white girl, aged 8 years, was first examined in the Section on Pediatrics. A diagnosis of cleidocranial dysostosis was made. In addition, bilateral coxa vara deformities of the hips were present. The mother of the patient consented to have roentgenograms made of her own thorax and hips.

She was found to have a condition similar to that of her daughter, except that there was very little, if any, coxa vara deformity of the hips. No roentgenograms were made to determine if the mother also might have cranial and dental abnormalities. A dental examination of the daughter was omitted at that time.

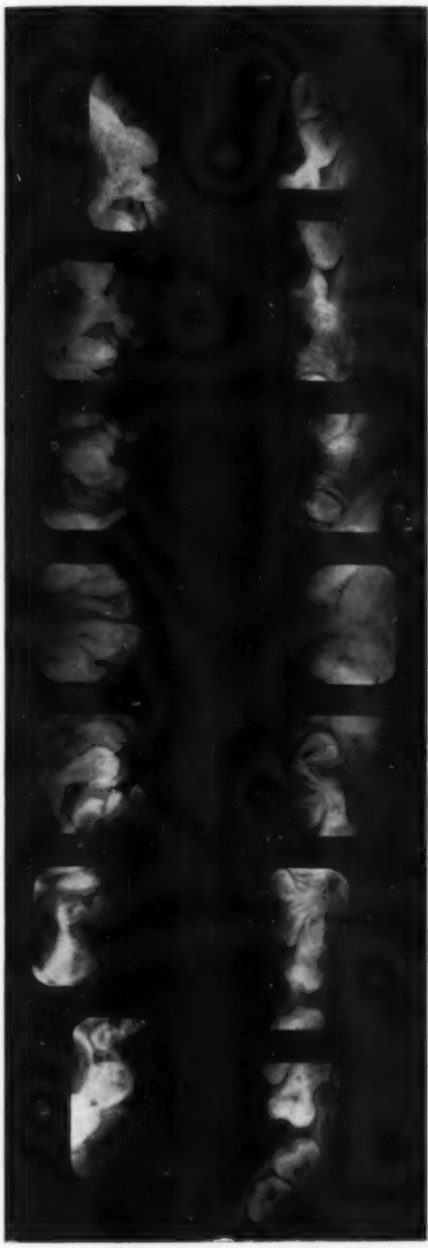

Fig. 6.—Roentgenograms made when the patient in Case 3 was 15 years of age. Some teeth which formerly were unerupted have erupted, and some of them have been removed. Orthodontic measures have failed. All of the permanent teeth are involved. Several supernumerary teeth are present.

When the patient was 13 years of age, she again returned to the clinic. Her chief complaint was that she had lost her deciduous upper and lower incisor teeth, and the permanent incisors had failed to erupt.

The deciduous canines and first and second molars were still present. Also present and in normal occlusion were the permanent first molars. This was interesting as there was a noticeable degree of prognathism. The maxilla was

closed and the palate was extremely high. It was noted that the lower lip was somewhat larger than the upper lip. Possibly the prognathism helped to emphasize the size of the lower lip. The alveolar ridges in the maxilla and mandible were quite broad and high.

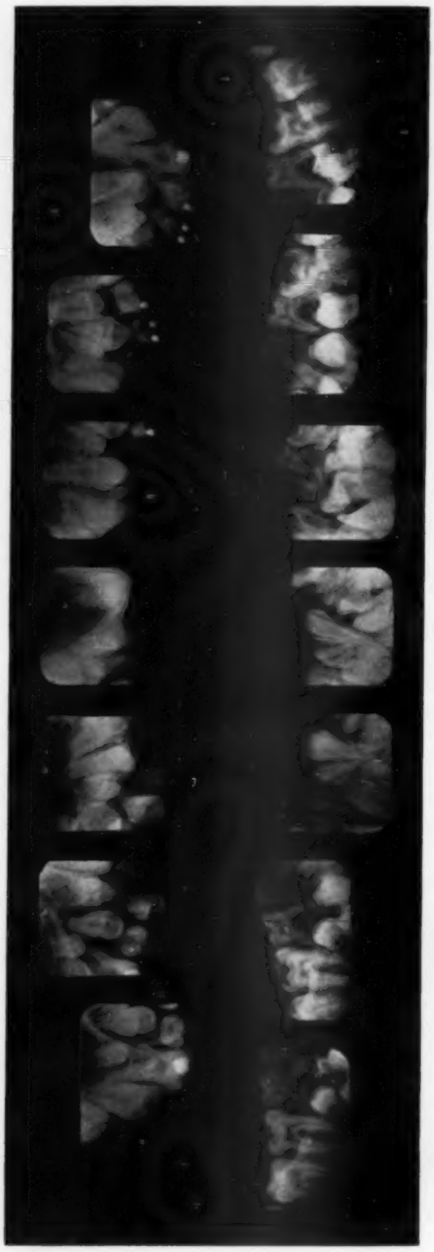

Fig. 7.—Dental roentgenograms in Case 4. Deciduous upper and lower incisors have been exfoliated normally, but the permanent incisors have failed to erupt. Permanent first molars have erupted and are in occlusion. The only teeth involved were the permanent teeth that followed deciduous teeth. There is practically one permanent supernumerary tooth for every normal permanent tooth.

Roentgenographic examination revealed many supernumerary teeth. In fact, by careful study, it appeared that, for every permanent tooth that one would ordinarily expect to find, there was also a supernumerary tooth (Fig. 7). Many of the incisors, especially the lower incisors, were misshapen and lying in an irregular design.

It would appear that possibly the deciduous upper left second molar would be exfoliated, and that this would be followed by normal eruption of the second premolar.

Laboratory tests did not disclose any abnormality.

COMMENT

It may be said that the dental findings in such cases are almost always constant. Initially, the deciduous teeth erupt normally and are retained longer.

All the permanent teeth may lose their eruption stimulus and stay embedded in the jaws as they did in Case 3. Again, only those permanent teeth that follow the deciduous dentition may be involved and fail to erupt, as in Case 4. This is apparently one of the bizarre dental peculiarities of this disease. There is no explanation for the lack or partial loss of eruptive stimulus of the permanent teeth, which is associated with partial failure of the phenomenon associated with resorption, and, finally, with shedding of the deciduous teeth.

Some of the permanent teeth may erupt, but the general tendency is for the permanent teeth to remain embedded in the jaws. Those persons who either have had their deciduous teeth removed, or have lost them and are wearing artificial dentures, will occasionally need to have permanent teeth removed. This may be due to the teeth finally erupting. More often there is resorption of the alveolar ridge, and as this occurs, the embedded teeth near the surface come into view. The question may well be raised, "Why not remove all of these embedded teeth before making dentures?" The answer to this question is that entirely too much of the alveolar process would need to be removed before these embedded teeth could be removed, as they are usually situated quite deep in the jaws. They are usually misshapen and irregular, hence more than a normal amount of bone would need to be removed. After this surgical procedure, the resulting alveolar ridge would not be too satisfactory for the wearing of artificial dentures. It follows that the favorable procedure is to remove these unerupted teeth if they make their appearance.

The high incidence of supernumerary permanent teeth, as well as the common occurrence of geminated teeth, is another of the dental peculiarities found in this disease. The hyperproductivity of the dental lamina to form supernumerary teeth is interesting, but difficult to account for. At the same time there is also a retarded growth of bone. In any event, the dental defect begins early in embryonic life.

The crowns of most of the permanent teeth appear to be of normal size, except where geminated teeth are seen to have developed. The crowns of geminated teeth are the monstrosities of the dental system. The roots of these unerupted permanent teeth show the most change, as they may be stunted or irregularly shaped. Also, there may be a tendency for these unerupted teeth to be irregularly arranged in the jaws. Occasionally, some may be inverted.

As would be expected, if there are many teeth present in the jaws, the alveolar ridges are quite thick and create the appearance of the gums being hypertrophied.

The maxilla is underdeveloped in comparison with the mandible, and has a micrognathous appearance. The roof of the mouth has a tendency to be quite narrow and high. There may be a tendency for failure of union of the two parts of the maxilla.

The mandible appears to complete its development as far as size is concerned. Although of normal size, the mandible appears to be prognathous. This appearance is the result of underdevelopment of the maxilla. This sometimes helps to emphasize a difference in the size of the upper and lower lips.

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IDIOPATHIC RESORPTION OF TEETH

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IDIOPATHIC resorption of teeth appears to attack the dentine primarily and may originate from either the inner or outer surface.

Several cases of idiopathic resorption of teeth have been reported in the literature. Authors have referred to this condition by various terms, such as "chronic perforating hyperplasia of the pulp," "pink spots," "internal granuloma," and "central osteoclastic resorption of dentine."

Miller,[5] in 1901, was one of the first authors to report such a condition. He reported a case in which extensive resorption of the walls of the pulp chamber had reduced the crown of the tooth to a mere shell. Subsequently, there was a redeposition of osteodentine of varying proportions, which imperfectly restored the lost tissue. It is of special interest that Miller said that, in 1890, Kirk had shown him an upper right central incisor in which absorption had taken place through the labial wall of the tooth until it had appeared immediately beneath the surface as a bright red spot. The thin wall of enamel had threatened to break through at any moment. In both of these instances the condition no doubt was the same as that which Mummery later referred to as "pink spots." These reports represent instances in which resorption of this type has produced extensive destruction of dentine in the coronal portion of the tooth.

The cause of this form of resorption is unknown. Several theories have been advanced as to the probable cause, and these have been considered by Soifer.[9] Mummery[7] reported one case in which a regulating appliance had been used, and another in which there was a history of a blow; in other cases there was no history of injury. He said that in many of the cases the bisymmetry of the affection pointed to some unknown etiologic factor. Cahn[2] said that by some unknown cause the pulp undergoes vascular changes which result in the resorption of the inorganic constituents of the dentine. Kronfeld[4] quoted Vandory as saying that a chronic inflammatory process in the pulp transforms it into granulation tissue, which, in turn, produces resorption of the walls of the pulp cavity. Thoma[11] expressed the opinion that an infectious process is not an etiologic factor since resorption takes place in teeth which are not carious and in which there is no pathologic lesion of the pulp or periodontal tissues. If infection is present, it is secondary to the resorptive process. Miller[5] said that cavities caused by resorption may be repeatedly found in the heart of the elephant's tusk, as well as in the teeth of the walrus where osteodentine is normally formed.

An explanation of the cause of the form of resorption which has its origin from the surface of the root was advanced by Hopewell-Smith,[3] who said that

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marginal gingivitis can result in the formation of granulation tissue and that this tissue can produce absorption of the hard tissues of the tooth. There are, no doubt, many instances in which resorption has its origin through the surface in the cervical portion of the root, and Applebaum[1] reported two cases of such resorption to substantiate Hopewell-Smith's theory. Mueller[6] reported a case in which there was extensive and rapid resorption in several teeth, and, from the illustrations shown, it appeared that the resorption in most of the teeth first involved the surface of the cervical portion of the roots.

The present report is based on a study of 179 cases of idiopathic resorption. Two hundred teeth were involved in the entire series of 179 cases.[10] The ages of the patients ranged from 19 to 76 years. Ninety-five of the patients were men and eighty-four were women. Although only a few of the 200 teeth were available for microscopic examination, many of them were observed roentgenographically from one to several years; therefore, it was possible to obtain information as to the degree and rapidity of the resorption.

TABLE I

SITE OF INVOLVEMENT IN 179 CASES OF IDIOPATHIC RESORPTION*

TEETH INVOLVED	SITE OF INVOLVEMENT	
	MAXILLA	MANDIBLE
Central incisor	45	44
Lateral incisor	15	11
Canine	18	14
Premolar	10	25
Molar		18
Total	88	112

*Two hundred teeth were involved in the entire series of 179 cases.

The site of involvement is shown in Table I. The lower teeth were involved more frequently than were the upper teeth, and the anterior teeth were involved more frequently than were the posterior teeth. This suggests that trauma might have been an etiologic factor as the anterior teeth are injured more frequently than are the posterior teeth. However, the average age of the patients was 48.2 years and patients of this age usually have more anterior teeth than posterior teeth.

No upper molar teeth were involved in this series of cases; however, it would be erroneous to conclude that these teeth do not undergo idiopathic resorption. The resorption may not be extensive; therefore, it may be overlooked in the dental roentgenogram. It does occur in the upper molar teeth, however, as Mummery[8] reported a case in which an upper second molar tooth was undergoing resorption.

In nineteen of the 179 cases in this series, more than one tooth was involved. In seventeen cases two teeth were involved, and in two cases three teeth were involved. In nine of the nineteen cases, adjoining teeth were involved. In four of the remaining ten cases, the resorption involved both upper and lower teeth.

In this series of cases, it was difficult to obtain accurate information regarding the etiologic role of trauma. People do not readily recall an injury of a tooth unless it has occurred rather recently. In some cases there was a history of trauma, and in all of these cases the injury had involved the incisor teeth.

In two cases there was evidence of fracture of the root of a central incisor tooth in the maxilla. In one of these cases the tooth responded to tests for vitality, but in the other the tooth did not respond to such tests. In each case the injured tooth had undergone resorption at a point distant from the line of fracture.

Only one of the 179 patients had worn an orthodontic appliance. This figure is not surprising, however, if one remembers that the average age of the patients was 48.2 years. When most of the patients were at the age at which orthodontic appliances ordinarily are worn, these appliances were not being used as extensively as they are at present. Resorption of the roots of teeth that results from orthodontic appliances has a characteristic form and is not easily confused with idiopathic resorption.

Sixty-two of the 200 teeth had been filled, probably because they had been carious. Eleven of these teeth, however, were serving as abutments for fixed bridges, and it is possible that some of these restorations originally had been placed on teeth that were free from caries. There were only three teeth with active caries and these had been filled previously.

One hundred sixty-five of the 200 teeth responded to tests for vitality. In several of those in which no response was obtained, the pulp chamber had markedly receded, and this may have accounted for a failure of the stimulus to reach the pulp. In others, the pulp tissue had been definitely destroyed by various causes. In a few cases it apparently had been destroyed by infection which had gained entrance through a perforation produced by the resorption. It was only in rare instances and in cases in which such perforation had taken place that patients gave a history of pulpitis. In by far the greater number of cases, the patients had not experienced any discomfort, even though the teeth had undergone extensive resorption.

The roentgenographic diagnosis of this type of resorption should not be difficult since any defect in the structure of the tooth which is not produced by caries must be the result of resorption of some kind. In cases of extensive resorption it is difficult to say whether the resorption originated within the pulp chamber or gained entrance from the outside by penetrating the cementum. From a review of many of the cases reported in the literature, and from a study of the roentgenograms in the series of cases under consideration, it appears that, in the majority of instances, the resorption begins on the root surface and extends inward. Applebaum stressed that a resorptive defect which appears to be confined to the center of the tooth in an anteroposterior roentgenogram actually may be found to be situated on, or communicate with, the outer surface of the tooth in a lateral roentgenogram. Fig. 1 shows anteroposterior and lateral roentgenograms of three incisor teeth and one canine tooth. These teeth had been extracted from the mandible before the roentgenograms were made. In the anterior views it appears as though the respective defects were confined largely to the center of the roots. The lateral views, however, reveal that the resorption is confined almost entirely to that portion of the root which is situated on the lingual side of the pulp canal and that there is a break in the surface of the root in each instance. Dental roentgenograms that have been made before extraction comprise a very unreliable method of determining whether resorption has originated within the pulp or on the surface of the root.

It is only in cases in which the defect is confined to the coronal portion of a sound tooth that is free of caries and in which there is no evidence of resorption beyond the cervical portion, that one can venture to assume that it originated from within.

Fig. 1.—Idiopathic resorption of three mandibular incisors and one mandibular canine. In the anteroposterior roentgenograms the resorption appears to be confined largely to the center of the roots but the lateral roentgenograms show that it is confined almost entirely to the portion of the roots on the lingual side of the pulp canal. A break in the root is visible in each instance. Roentgenograms were made after the teeth had been extracted.

Fig. 2.—Extensive resorption of dentine in an upper second premolar. A layer of unresorbed dentine may be seen surrounding the pulp chamber.

The dentine adjacent to the pulp, as well as the cementum, is very resistant to resorption and often remains unresorbed. Careful examination of roentgenograms often reveals evidence of a thin layer of unresorbed dentine; in some instances, this dentine produces radiopaque lines that frame the pulp chamber and pulp canals with striking clearness. Such evidence is present in a roentgenogram of an upper second premolar tooth which has undergone resorption (Fig. 2). In the roentgenogram, the pulp appears to be completely surrounded by dentine, a finding which lends one to question whether the resorptive process has had its origin from the pulpal wall, even though resorption has been confined almost wholly to the coronal portion of the tooth. Clinical examination did not reveal any defect on the exposed surface of the tooth. The enamel pre-

Fig. 3.—Photomicrograph of longitudinal section of upper lateral incisor shown in insert; *a*, cementum; *b*, dentine; *c*, osteoid tissue; *d*, connective tissue; *e*, opening in cementum where resorption may have originated (×20).

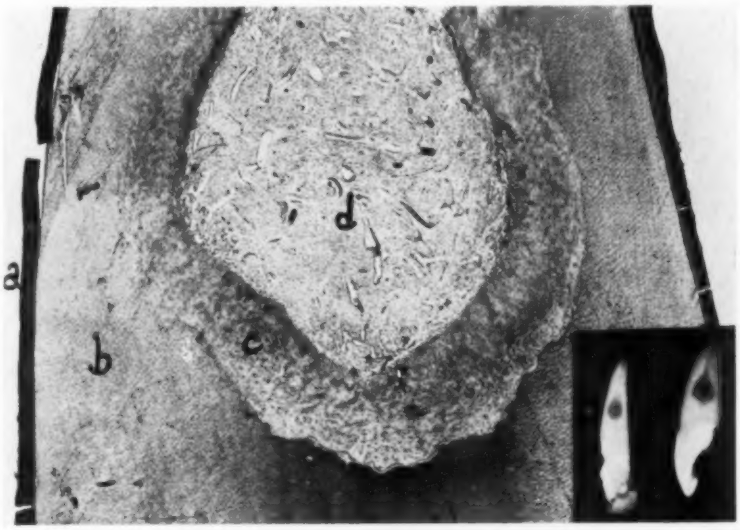

Fig. 4.—Photomicrograph of longitudinal section of upper lateral incisor shown in insert; *a*, cementum; *b*, dentine; *c*, osteoid tissue; *d*, pulp (×14).

sented a pinkish hue, owing to the highly vascular tissue which is immediately beneath it. There was a normal response to tests for vitality, and the tooth had at no time been painful.

Anterior and lateral roentgenograms of two extracted upper lateral incisors are shown in inserts in Figs. 3 and 4. These teeth were removed from the same patient. An opening on the surface of the root can be clearly seen in both roentgenographic views of the tooth shown in Fig. 3. Longitudinal microscopic sections of this tooth revealed a definite opening on the palatal surface of the root. The resorptive process may have had its origin at this point. No opening could be seen on the labial surface in several microscopic sections that were made. It also revealed that the resorbed dentine had been almost completely replaced by osteodentine (Fig. 3). It is at once apparent that osteodentine is in no measure as radiopaque as dentine since the limits of the entire region in which dentine has been resorbed are clearly visible in the roentgenogram.

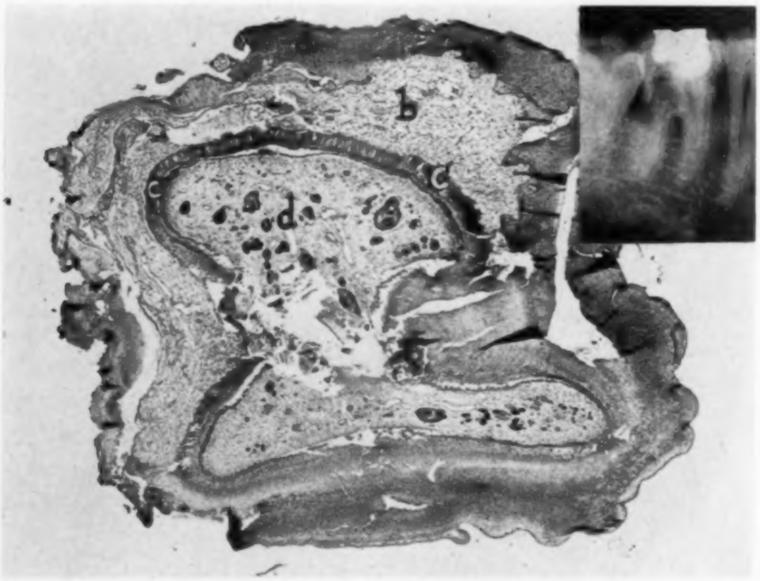

Fig. 5.—Photomicrograph of transverse section of distal root of first molar shown in insert; *a*, dentine; *b*, resorption of dentine: *c*, layer of unresorbed dentine surrounding pulp; *d*, pulp (×12).

In the anteroposterior and lateral roentgenogram of the lateral incisor tooth shown in the insert in Fig. 4, there is no evidence of an opening on the surface of the root, and it appears that the resorptive process has been confined to the center of the root. This was verified by longitudinal microscopic section (Fig. 4). The pulp chamber of the tooth was obliterated, and the pulp canal was markedly enlarged in the region of the resorption. Here again a large portion of the resorbed dentine had been replaced by osteodentine.

A roentgenogram of a lower first molar tooth that had undergone resorption is shown in the insert in Fig. 5. At the time the roentgenogram was made, the patient complained of pain in this tooth and said that the pain had been present for two days. Clinical examination revealed a region of softening on the distal surface of the distal root slightly below the gingival margin. The tooth was

extracted and microscopic sections were made. The section in Fig. 5 is a transverse section of the distal root made at a point near the bifurcation of the roots. There was extensive resorption of dentine and the resorbed dentine had been replaced by a loose connective tissue. The pulp remained intact and was surrounded by a layer of dentine of uniform thickness. In some of the sections there was evidence of the formation of osteoid tissue.

Material for microscopic examination was available in only seven cases. This material consisted of a total of ten teeth: two lower molars, two upper lateral incisors, one upper central incisor, four lower incisors, and one lower canine. There was evidence of typical resorption of the hard structure of all of the ten teeth.

In twenty-one cases, the involved teeth were observed roentgenographically from one to thirteen years. In two of these cases the patients had two teeth that were involved; therefore, twenty-three teeth were involved in the twenty-one cases. In seven cases, dental roentgenograms had been made before the onset of resorption. In some of the teeth the resorption appeared to have been arrested; in others it had progressed. Only one tooth had been lost as a result of the resorption.

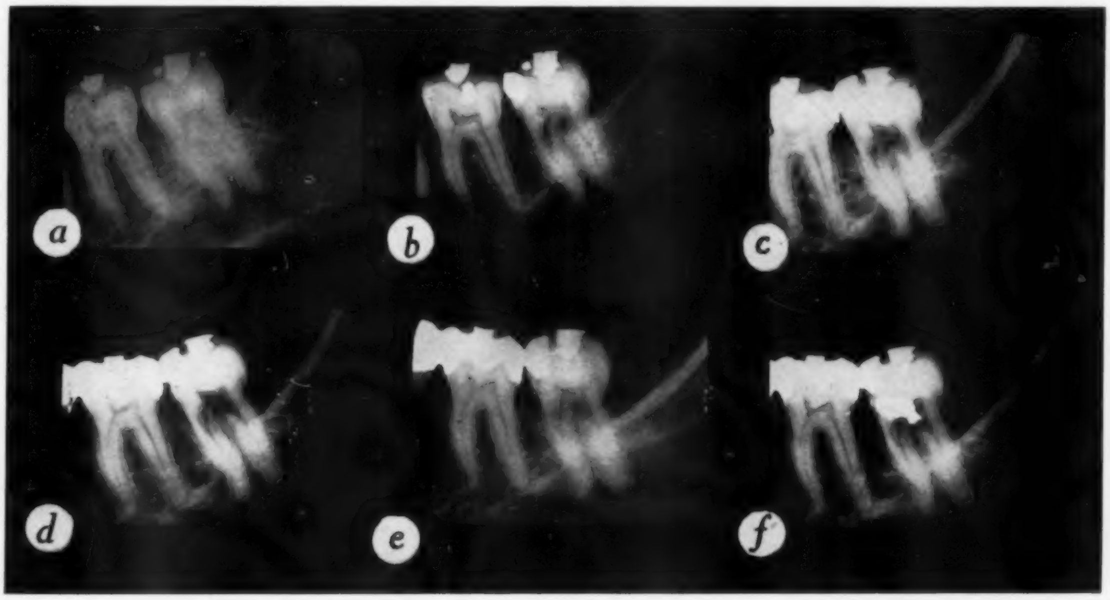

Fig. 6.—Roentgenograms showing progress of resorption of a lower second molar; *a*, made in 1930; *b*, made in 1931; *c*, made in 1933; *d*, made in 1935; *e*, made in 1937; *f*, made in 1941.

In several of the 179 cases there were interesting features. The following case is an example.

A woman, aged 20 years, who came to the clinic in 1931, had a lower right second molar tooth that had undergone resorption. Roentgenograms that had been made in 1927 and on Nov. 7, 1930 (Fig. 6*a*), had not disclosed any evidence of resorption of this tooth. The roentgenogram shown in Fig. 6*b*, which was made on Sept. 24, 1931, was the first one that disclosed resorption of this tooth. It is of interest to note that rather pronounced resorption occurred in the in-

terval between Nov. 7, 1930, and Sept. 24, 1931. An additional filling had been inserted in this interval, but in view of the many instances in which resorption occurs in unfilled teeth, one would not be justified in concluding that the resorption was caused by dental caries in this case. The roentgenograms shown in Fig. 6*c*, *d*, *e*, and *f* were made in 1933, 1935, 1937, and 1941, respectively. It appears that the maximal destruction occurred in 1935; thereafter, it is possible that deposition of osteodentine kept pace with the resorptive process. Possibly one may be justified in this assumption in view of what occurred in the interval between 1937 and 1941. In 1939, in the course of a periodic dental examination, the patient's dentist discovered a painless soft spot on the buccal surface of the tooth, near the cervical margin of the crown. In preparing a cavity for filling, it was found that solid normal dentine could not be reached and the dentist realized that he was not dealing with the usual carious process. An amalgam filling was inserted, and the prognosis for continued vitality of the pulp was considered poor. When the tooth was examined in January, 1941, its color was normal and it responded to tests for vitality. The filling may have been placed in osteodentine and it is possible that the tooth may be retained indefinitely.

TABLE II

INCIDENCE OF SYSTEMIC DISEASE*

SYSTEMIC DISEASE	IN 179 CASES OF IDIOPATHIC RESORPTION OF TEETH		IN 100 CONSECUTIVE CASES IN WHICH PATIENTS WERE REFERRED TO THE DENTAL SECTION
	NUMBER	PER CENT	NUMBER AND PER CENT
None demonstrable	58	32	36
Degenerative arthritis	30	17	15
Fibrosis and rheumatoid arthritis	24	13	14
Glandular dysfunction	7	4	2
Gastrointestinal disease	14	8	9
Cardiovascular disease	14	8	7
Neurologic disease	16	9	5
Genitourinary disease	10	6	6
Dermatologic disease	9	5	3
Tumors	7	4	4
Respiratory infection	5	3	9
Allergic disturbances	4	2	4
Syphilis	4	2	1

*More than one systemic disease was present in several cases in each group.

A complete medical examination was made in each of the 179 cases of idiopathic resorption. The incidence of systemic disease in these cases and in a control series of 100 patients who were sent to the dental section for examination is shown in Table II. There is no evidence that idiopathic resorption of teeth is secondary to, or associated with, any particular systemic disease. In 32 per cent of the 179 cases of idiopathic resorption and in 36 per cent of the control series, the patients were healthy and normal. The incidence of degenerative arthritis (osteoarthritis; hypertrophic arthritis) and the incidence of fibrositis and rheumatoid arthritis were very nearly the same in the two groups of cases. The high incidence of arthritis can be accounted for by the fact that patients with arthritis are more frequently referred to the dental section for

examination than are patients who have other diseases. Endocrine dysfunction was present in seven, or 4 per cent, of the 179 cases. The nature of the endocrine dysfunction was as follows: nontoxic adenoma of the thyroid gland in three cases, exophthalmic goiter in one case, diabetes mellitus in two cases, and Addison's disease in one case. In none of the 179 cases was there any clinical or roentgenographic evidence of parathyroid disease.

SUMMARY AND CONCLUSIONS

The origin of idiopathic resorption is, in the majority of instances, from the surface of the root. At first the resorption may be very rapid, but later it may continue slowly for a long time, and in many instances it may become arrested. The osteodentine which is formed is not as radiopaque as the normal dentine which it replaces; therefore, a resorptive defect will always be clearly visible in the roentgenogram even when there is no longer any active resorption and even though the resorbed dentine has been completely replaced by osteodentine. From roentgenograms and microscopic sections studied it is apparent that resorption tends to progress through dentine parallel to the long axis of the tooth; that part of the dentine proximal to the pulp and cementum is more resistant and is destroyed only when resorption is extensive. Resorption apparently is not associated with any particular systemic disease.

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BASAL METABOLIC RATES AND DENTAL CARIES

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RECENTLY, during the physical examination at the Mayo Clinic of a group of nurses who were being admitted for training to the Kahler Training School, a dental examination with dental roentgenograms was included, as well as a determination of the basal metabolic rate. The incidence of dental caries appeared to be slightly greater among those women whose basal metabolic rates were on the lower side of normal than among the rest of the group.

There were forty-three women in this group. Basal metabolic rates, which were obtained for twenty-eight, varied between plus 6 and minus 24 per cent. The patients were divided into groups according to their basal metabolic rates. The incidence of caries was progressively greater as the basal metabolic rate for the different groups decreased. The incidence of carious teeth, both primary and total, was compared with the rates. As the number of carious teeth increased, there was an associated decrease of the basal metabolic rates. Since the rates for this first group of nurses were not obtained for all members of the entering class, it seemed advisable to repeat the study on the next incoming class of nurses and to obtain rates for all of them.

Accordingly, the next group of sixty-three nurses were examined and their carious teeth charted; then their basal metabolic rates were determined. It was noted that in comparing the average basal metabolic rates with the incidence of caries, and the incidence of caries with average basal metabolic rates, there is the same tendency as in the first group: namely, a correlation between dental caries and a progressively decreasing low normal basal metabolic rate. The basal metabolic rates of both groups of student nurses were on the average somewhat below the Mayo Foundation standards; the first group ranged from

TABLE I

DENTAL CONDITIONS ACCORDING TO BASAL METABOLIC RATE

BASAL METABOLIC RATE (PER CENT)	CASES	CARIES		TEETH		
		TOTAL	PRIMARY	FILLED	PRESENT	EXTRACTION ADVISED
+11 to - 4	86	2.3	1.6	9.6	30.0	1.5
- 5 to -10	73	2.6	1.9	9.6	29.7	1.9
-11 to -24	73	3.3	2.5	9.9	30.4	1.9
Total	232	2.7	2.0	9.7	30.0	1.8

TABLE II

BASAL METABOLIC RATE ACCORDING TO DENTAL CARIES

TOTAL CARIES	CASES	AVERAGE BASAL METABOLIC RATE (PER CENT)	PRIMARY CARIES	CASES	AVERAGE BASAL METABOLIC RATE (PER CENT)
0- 2	123	-5.7	0- 2	160	- 6.1
3- 6	91	-7.4	3- 6	68	- 7.8
7-10	18	-9.1	7-10	4	-11.5

Section on Dental Surgery, Mayo Clinic.

plus 6 to minus 24 per cent, and the second group from plus 4 to minus 22 per cent. Discussion of this fact, however, will be deferred, as it involves the entire problem of standard values for basal metabolism.

The two succeeding classes of nurses were reviewed and appeared to show the same tendency. All groups were combined and the date summarized in Tables I and II. The number of patients is still too small to permit definite conclusions; yet the evidence that a relationship does exist between these conditions is sufficient to justify further studies. Further evidence of this relationship is shown in Fig. 1. The average basal metabolic rate was obtained for each unit of dental caries. The number of cases used in obtaining each average is indicated above each point. A straight line was fitted to the points and the equation is given in Fig. 1. From the equation one notes that for each additional carious tooth the basal metabolic rate decreases approximately half a unit, on the average.

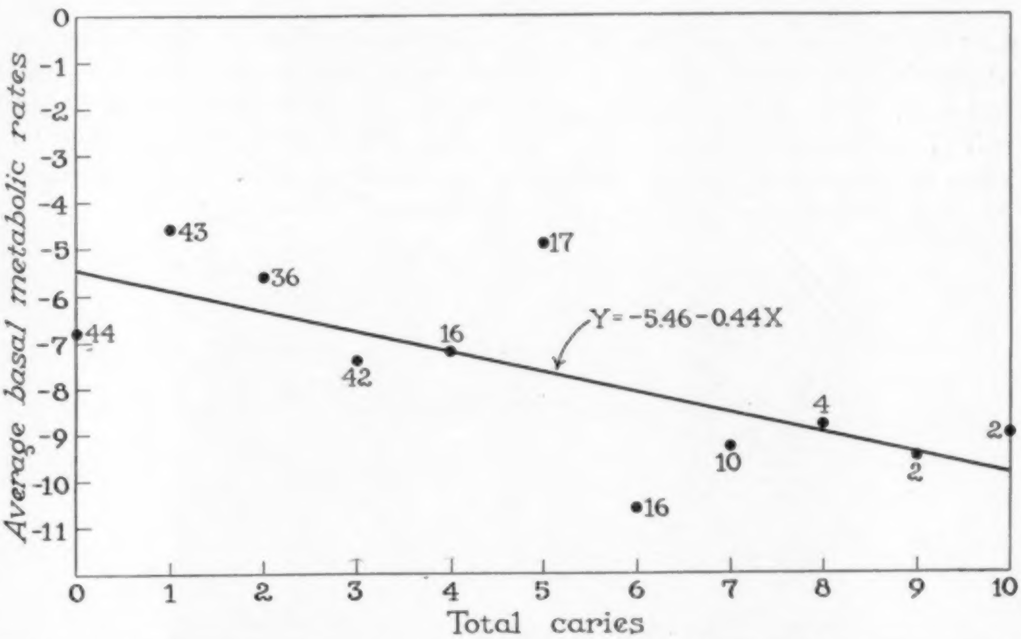

Fig. 1.—Relation of average basal metabolic rate to total dental caries. Numbers represent number of persons having the stated number of carious teeth.

Groups of young women entering upon a nursing career are ideal for this type of study, as they are of uniform age, 18 to 21 years, and are in excellent health as shown by general physical examination. Also, from the dental aspect the group is of advantage, as few of their teeth have been lost at this age and their general oral condition is good. The use of the dental roentgenogram permits a review of their caries at any time, so that later studies of progress can be made.

Primary caries, spoken of in the tables, consists of those cavities appearing at the contact points that do not show any evidence of having had a previous filling or restoration. Secondary caries consists of those cavities that appear at the margins of a filling, showing that an attempt has been made to restore the carious tooth.

DECALCIFICATION OF THE CROWNS OF TEETH IN SITU

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West and Judy have studied the destruction of dental enamel by acidified candies and placed particular stress upon the importance of citric acid. They stated that the occasional use of such "fruit drops" would cause little harm; on the other hand, the continued and habitual use of such candy is likely to result in serious damage to dental structure.

REPORT OF A CASE

With a history of pain in the left tonsillar region of fourteen months' duration, a white man, 62 years of age, on examination by the Section on Laryngology, Oral and Plastic Surgery of the Mayo Clinic, presented a large firm mass which involved the left tonsil, the base of the tongue, and the lateral wall of the hypopharynx. Biopsy of a specimen from the tongue revealed the lesion to be a squamous cell epithelioma, Grade 3 (Broders' method). Irradiation was prescribed and the patient returned home for treatment.

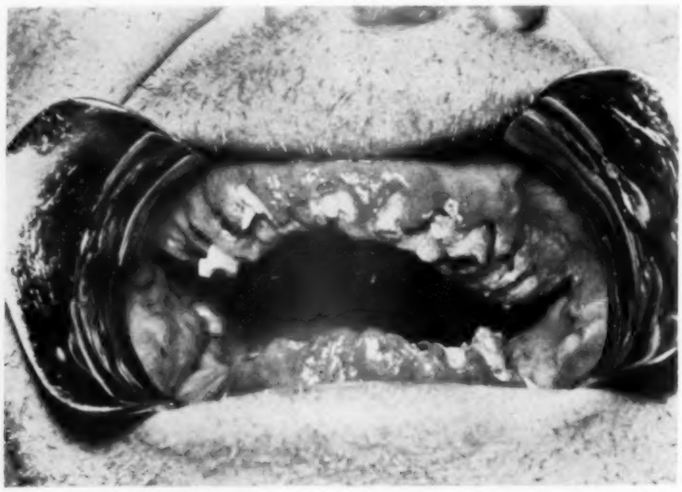

Fig. 1.—Decalcification due to use of cough drops.

Nine months later the patient came back to the clinic and was found to be in good general condition with no evidence of recurrence of the epithelioma. At that time he was seen in the Section on Dental Surgery. The results of dental examination and the roentgenograms were quite normal, with a molar tooth in the left side of the mandible being indicated for removal. There was no clinical or roentgenographic evidence of dental decalcification.

A year later, a letter from this patient stated that shortly after his last visit to the clinic he had had three teeth removed, followed by considerable post-

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operative trouble. He complained that his teeth had "gone to nothing" and asked regarding the advisability of removing all of his teeth.

Six weeks after the arrival of the foregoing letter, the patient registered at the clinic for the third time, thirteen months having elapsed since the second visit. The results of dental examination at this time were as follows: The

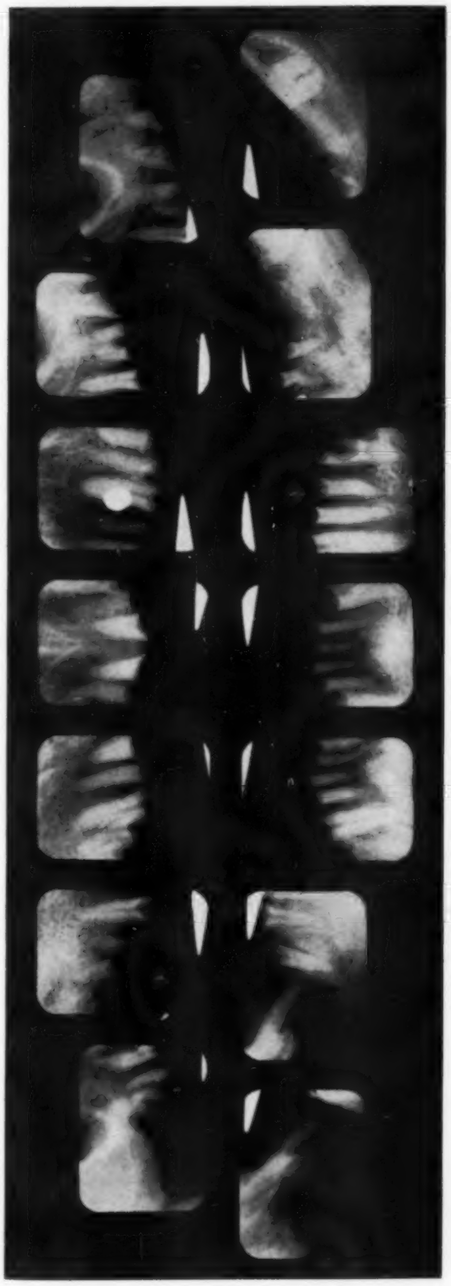

Fig. 2.—Roots normal, crowns decalcified.

breath smelled strongly of creosote. The entire crowns of all the teeth had become completely decalcified (Fig. 1). The portions of teeth still visible were flexible and rubbery, comparable to dental substance following its complete decalcification in nitric acid. Pain was not present. The chief complaint was lack of function. A large area of leucoplakia was found on the cheek and sur-

rounding structures in the region of the right retromolar triangle and the commissures of the jaws. The crowns of the teeth had been decalcified so completely that they were no longer discernible in the roentgenogram (Fig. 2) and clinical examination revealed that the enamel of all the teeth had been destroyed.

The twelve mandibular teeth, such as they were, were removed while the patient was under local anesthesia, and ten days later the fifteen remaining maxillary teeth were also removed. No complications were experienced.

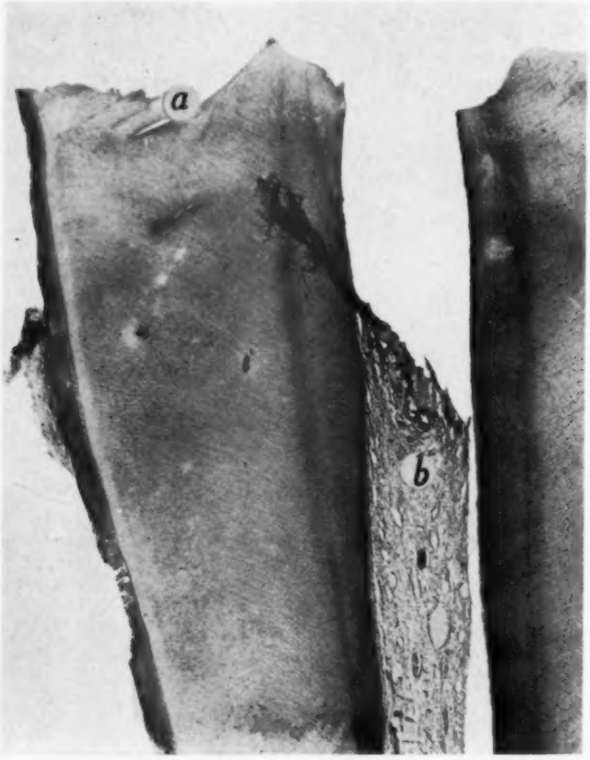

Fig. 3.—Section of decalcified tooth showing *a*, absence of crown, *b*, pulp with localized inflammatory reaction (hematoxylin and eosin ×17).

The extracted teeth were not unusual, except for the lack of coronal structure and their strong odor of creosote. The line of demarcation between the normal root and the decalcified portion of the tooth was abrupt. The dental pulp of a maxillary canine tooth showed a localized inflammatory reaction restricted to that part of the pulp nearest the crown, with no reaction evident in the remainder. No degeneration was present (Fig. 3).

COMMENT

Within an interval of thirteen months all of these teeth had undergone decalcification. The picture is not that of caries occurring in a dry mouth. The explanation may rest with the constant use of cough drops which the patient had found necessary to relieve the xerostomia which followed irradiation therapy. Chewing gum, different types of candies and various cough drops had been tried in an effort to stimulate the secretion of saliva. The cough drops that the patient finally selected and used continuously (he said that he always had one

in his mouth, a fact which may also account for the leucoplakia) contained as active ingredients menthol, beechwood creosote, eucalyptol, horehound, white pine, and wild cherry.

It has been found that when one of these cough drops is dissolved in 100 c.c. of water, a solution of pH 5.72 to 6.2 is obtained. There was some variation between the different cough drops in the same package and also between the cough drops in different packages.

The remarkable features of this case are (1) the limitation of the process of decalcification to the exposed tooth structure in situ, and (2) the relative lack of involvement of the dental pulps, and the freedom from pain.

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Editorial

The Mayo Number

This month we present a collection of papers from the Mayo Clinic. Various departments of the Clinic have made contributions, namely, the sections on Dental Surgery, Laryngology, Oral and Plastic Surgery, Otolaryngology and Rhinology, and the Division of Medicine. Cooperation between the dental department and other departments is of great importance in any clinic, and the material presented herewith shows that, in the Mayo Clinic, as in most prominent clinics and hospitals, the various departments work together for the benefit of the patient.

Frequently, patients are seen in private practice who should have consultations with specialists in the different and varied fields of medicine; unfortunately, such consultations are not always as freely recommended in an office as they are in the clinics where, patients can be seen quickly by men in other specialties working in the same establishment.

Many complimentary letters have been received in response to the Massachusetts General Hospital Number. Therefore, in 1944, we are going to devote several issues to the presentation of material from some of the well-known clinics in the United States.

K. H. T.

Erratum

In the article entitled ''The Dentist's Responsibility in Oral Tumors'' by W. N. Burford, which appeared in the November, 1943, issue of the JOURNAL, volume 29, page 612, the first sentence should read: ''Oral malignancies account for approximately 3 per cent of deaths caused by cancer.''

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Original Articles

PERTINENT FACTS ABOUT THE TONGUE AND BREATH AS AIDS IN THE DIAGNOSIS OF DISEASE

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THE future of dentistry as one of the important professions is liable to be somewhat in the balance if all the members of the profession do not equip themselves with the knowledge that will make them "specialists" of the mouth and all that lies therein. The teeth, the gums, the periodontal membrane, the alveolar bone, the mucous membrane, the cheeks, the hard and soft palate, the pharynx, and the tongue are all important constituents of the mouth, and, therefore, should be treated by the dentist. There are still many dentists, however, who fail to realize that the scope of dentistry is much wider than the filling and extraction of teeth and the restoration of lost teeth by prosthetic appliances. It is imperative that we realize now that the mouth is the dentist's domain, for if we do not, then it is entirely possible that the diagnosis and treatment of oral disease will become a medical specialty in the hands of physicians, and dentistry will revert toward its former position of a purely mechanical vocation.

Two of the most important oral constituents that have been sorely neglected by the profession are the tongue and the breath. Besides being important indicators of oral disease, the color and appearance of the tongue and the odor of the breath are pathognomonic of various systemic conditions. It is interesting to note how well informed about the tongue and breath were the ancient members of the healing arts; as a matter of fact, they were so well informed that they often were able to diagnose general pathology from a mere inspection of the tongue or the breath, or of both. There are innumerable cases and records to this effect, but it seems as if this ability is becoming one of the lost arts of the medicodental profession. This is quite understandable when we think of the important role the sciences of bacteriology and roentgenology, in particular, are playing in the diagnosis of disease. So important has been this role that we find that a tendency is arising to depend exclusively on these two sciences in diagnosis. The fallacy in this is obvious when it is remembered just how many pathologic conditions are not evident in a radiograph, and the innumerable conditions in which the results of bacteriologic investigation alone are not enough to provide any definite diagnosis. True, these are important factors in diagnosis, but so are those signs and symptoms that are evident in the organs of the special senses if we are willing and able to interpret these when present. It makes for a much more reliable diagnosis, then, if we employ all available evidence, whether bacteriologic, radiographic, or of the special senses, as aids in the diagnosis of disease instead of considering any one alone to be diagnostically

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conclusive. It is being suggested that we retrieve some of the art of diagnosis by using the appearance of the tongue and the odor of the breath as aids. Furthermore, this should be the function of the dentist, and, obviously, it will be helpful to him in assisting the physician, thus resulting in the cooperation so definitely needed between the two professions.

The tongue is the principal organ of the sense of taste and an important organ of speech. It assists in the mastication and deglutition of food. Situated in the floor of the mouth within the curve of the body of the mandible, it presents a root, apex, inferior surface, and dorsum. The tongue is connected by its root with the hyoid bone, the epiglottis, the soft palate, and the pharynx. The apex, dorsum, and inferior surface are free. The dorsum of the tongue is covered with papillae, which are thickly distributed over the anterior two-thirds, giving this surface its characteristic roughness. These papillae are divided into:

1. Papillae vallatae, which are large and from eight to twelve in number. Each papilla is shaped like a truncated cone, the smaller end being directed downward and attached to the tongue, the base projecting a little above the surface of the tongue and studded with numerous small secondary papillae and covered by stratified squamous epithelium.

2. Papillae fungiformes, which are more numerous and are formed chiefly on the sides and apex, but are scattered irregularly and sparingly over the dorsum. They can be differentiated from the other papillae by their large size, rounded eminences, and deep red color. They are narrow at their attachment to the tongue, but broad and rounded at their free extremities, and covered with secondary papillae.

3. Papillae filiformes, which cover the anterior two-thirds of the dorsum. Minute and threadlike, these papillae are arranged in lines parallel to the two rows of papillae vallatae, except at the apex of the organ where their direction is transverse. Projecting from their apices are whitish secondary papillae. The color is due to the thickness of the epithelium of which they are composed. The cells of this epithelium are keratinized and elongated into dense brushlike processes. The larger and longer papillae of this group are termed papillae conicae.

4. Papillae simplices, which are similar to the skin and cover the whole of the mucous membrane of the tongue in addition to covering the larger papillae. They consist of microscopic elevations, each containing a capillary loop and covered by a layer of epithelium.

The tongue contains a number of muscles which are supplied by the hypoglossal nerve and lingual artery. It is partly invested by mucous membrane and a submucous fibrous layer. The mucous membrane differs in different parts. Covering the inferior surface is a thin smooth membrane similar to that lining the rest of the oral cavity, and on the dorsum is a thick covering freely movable over the subjacent parts. This contains a large number of lymphoid follicles, each follicle forming a rounded eminence, the center of which is perforated by a minute orifice leading into a funnel-shaped cavity. Around this cavity are grouped numerous oval nodules of lymphoid tissue, each enveloped by a capsule derived from the submucosa. The mucous membrane on the anterior part of

the dorsum of the tongue is thin and closely adherent to the muscular tissue. The tongue is provided with mucous and serous glands. The former are found especially at the back of the tongue in the region of the taste buds which are the end organs of the gustatory sense, but are also present at the apex and marginal parts.

The tongue is normally moist and pink in color but may become discolored from the local effects of food or other substances. Foods which, when ingested, cause discolorations of the tongue are blueberries, blackberries, licorice, red wine, colored candy, etc. Certain drugs, coffee, tobacco, laudanum, iron compounds, and medicines also produce temporary discolorations. In addition to these purely local and nonpathologic causes, the tongue becomes discolored and loses some or all of its normal characteristics when it becomes involved in disease. Disease may attack the tongue itself or may spread to it from immediate adjacent parts; for example, the lips, cheeks, mucous membrane, pharynx, etc. Systemic diseases may also affect the tongue, producing abnormal changes.

Table I

Congenital Defects of the Tongue

a. *Aglossia.*—A congenital absence of the tongue due to faulty embryonic development
b. *Bifid* or *Cleft Tongue.*—A congenital anteroposterior splitting of the tongue. It sometimes occurs in combination with other facial clefts
c. *Congenital Absence of Papillae.*—Here the filiform papillae and fungiform papillae of the tongue are either completely or partially absent
d. *Fissured Tongue.*—This is also called lingua plicata or scrotal tongue. Here the tongue is larger than normal causing the mucosa to become plicated. The depression in the median line is extremely deep, and in addition other deep furrows are seen over the entire dorsum. The tongue shows no induration and is soft and pliable. Some cases are acquired. The condition persists throughout life
e. *Lobulated Tongue.*—A congenital presence of an extra lobe lying directly on the tongue
f. *Long Tongue.*—A few instances of extreme length of the tongue have been recorded
g. *Lymphangiomatous Macroglossia.*—Often congenital. Progressive enlargement of tongue, with small vesicles appearing on the surface. Tongue may protrude from the mouth
h. *Microglossia.*—Abnormally small tongue.
i. *Partial Ankylosis.*—Also called "tonguetie." Here there is a shortened frenum beneath the tongue, resulting in hindered mobility and protrusion, and indistinct speech

The breath consists of the air taken in and expelled by the expansion and contraction of the thorax. The healthy mouth should be free from any offensive odor. Offensive breath in various forms occurs as a temporary or permanent disturbance, and often may be diagnostic of some disease condition. Prinz has given the following classification:

1. Odors arising from purely dental conditions. Ninety per cent of all cases originate from prolonged stagnation and bacterial fermentation of food debris about the teeth. A sickly foul odor is produced from carbohydrates, ammoniacal compounds, and from hydrogen sulfide. Prosthetic appliances may be covered with mucoid deposits which undergo fermentation and produce offensive odors.

TABLE II

LOCAL DISEASES AFFECTING THE TONGUE AND BREATH AND DISEASES OF THE TONGUE ITSELF

CONDITION	ETIOLOGY	APPEARANCE OF TONGUE
ACTINOMYCOSIS	Infection by *Streptothrix actinomyces* entering through the mouth, carious teeth, and gums	First a painless small lump appears on the tongue. This increases to a pale yellow swelling which may break and discharge pus containing sulfur granules in which fungi are found. The whole tongue becomes hard, and difficult to move
ACUTE DIFFUSE GLOSSITIS	Injury caused by mechanical and chemical agents, stings from insects, poison ivy, vulcanite plates	Abscesses form on the tongue, which is red, swollen, and tender. Pain on mastication and edema extending to the fauces are associated symptoms
BLACK HAIRY TONGUE	Streptothrix yeasts and chromogenic bacteria penicillium (micor niger) cause a thick matting	There is hypertrophy of the filiform papillae with intensification of their color. The condition begins in a small area of the dorsum. This tends to spread and recur after removal. The filiform papillae can be removed and appear as long filaments, resembling hair. The tongue resembles a matted black fur
BURNING TONGUE	The local factors include sharp cusps, rough margins on fillings, galvanic currents due to dissimilar metals. Anemia, disturbances of gastric secretion, and psychoneurosis are among the systemic factors	There are complaints of burning sensations of the tongue
CARCINOMA	It may arise from a papilloma, advanced leucoplakia, or without preceding pathologic conditions	Epidermoid carcinoma forms on the sides and dorsum. It may be molluscous or papillomatous. In other cases there is only a small surface ulcer with induration. Sometimes the lesion is buried in the base of the tongue with little external evidence. Pain is the first symptom. Later the tongue causes awkwardness in eating and speaking. Adenocarcinoma forms in the apical gland region on the lingual surface. There is a lumpy feeling in the tongue
CHRONIC RECURRENT ULCER (APHTHAE)	Disturbances of internal secretion	Small vesicles appear on the tongue. These increase in size, forming small painful ulcers surrounded by a red margin. There is a glossitis
DECUBITAL ULCER	Injury by a ragged tooth or poorly fitting dentures	The ulcer is definitely outlined, with an indurated border presenting a yellow-gray exudate on the surface. The border may become thickened and form a crater
GEOGRAPHIC TONGUE	Unknown	The condition starts as a white spot which heals on one side and leaves a white crescent-shaped defect surrounding red denuded patches. These may spread or coalesce with others, and are the results of desquamation of epithelium on the dorsum
LEUCOPLAKIA	Excessive use of tobacco in chewing and smoking. Other predisposing factors include spicy foods, alcohol, oral sepsis, poorly fitting dentures, and syphilis	There is a glistening white, hard, rough thickening of the epithelium on the dorsum. In advanced cases, cracks are liable to occur, and movement of the tongue is hindered

TABLE II—CONT'D

CONDITION	ETIOLOGY	APPEARANCE OF TONGUE
LICHEN PLANUS	Skin disease. Etiology unknown	On the dorsum of the tongue are found smooth white patches which are not hard
LUDWIG'S ANGINA	Streptococcus infection of the floor of the mouth	The tongue may be swollen. An offensive breath is evident
MOUTH BREATHERS	Nasal obstruction	The tongue is white, coated, and dry. The breath is very offensive
MUCOUS CYSTS	Occlusion of the orifices of the ducts from the anterior glands of the tongue	There are swellings under the surface of the tongue which extend as bluish-green vesicles which are soft and fluctuant
NEUROTROPHIC ULCER	Injury of the peripheral nerves	Erosions appear on the tongue, which has a grayish surface with an undetachable membrane. This may be covered by a fibrinous exudate of yellowish color. The tongue may be painful
PARESTHESIA	Removal of several teeth or extensive oral surgery	There is numbness, burning, itching, tingling, prickling, or coldness of the tongue
PERIODONTOCLASIA	Local and systemic factors	The tongue is coated and the breath offensive
PHARYNGITIS	Oral sepsis	The tongue is yellowish gray and furred. The breath is offensive
SPRUE	Associated with food deficiency diseases and exhaustion	The epithelial surfaces of the tongue are denuded in irregularly shaped patches. The denuded areas are painful and become ulcerated in advanced cases, and are covered with a yellowish slough around the edges
STOMATITIS	The etiology of a simple noninfectious stomatitis may be chemical, mechanical, or thermal	The tongue is furred and yellowish gray in color. The breath is offensive
SYPHILIS	Spirochete of syphilis	Chancres of the tongue are the nonulcerative deep-seated types of lesions, which are indurated and covered with a firm mucous membrane
THRUSH	Infection by monilial organisms	There are white patches on the surface of the tongue, which is red, smooth, and sensitive to hot fluids and tobacco
TONSILLITIS	Oral sepsis resulting in the infection of the tonsils	The tongue is coated and breath offensive
TROPHIC ULCER	Local use of anesthetics	A blood blister may occur on the tongue, then desquamation of the epithelium takes place, followed by necrosis causing a circular depressed surface ulcer with grayish fundus
TUBERCULOSIS OF THE TONGUE	Infection by the tubercle bacillus of a superficial wound	An ulcer is seen on the tip and sides of the tongue where carious teeth have caused abrasions. The superficial ulceration is shallow, irregular in shape, violet in color, and with festooned borders
TUMORS OF THE TONGUE: ANGIOMA, PAPILLOMA, LIPOMA, SARCOMA, FIBROMA	Irritation, etc.	The tongue is swollen
VINCENT'S GLOSSITIS	Infection by Vincent's organisms	The tongue is covered by a detachable membrane on an inflamed surface. When the membrane is pulled away, a bleeding irregular surface is seen. There is a metallic taste and the breath is fetid

TABLE II—CONT'D

CONDITION	ETIOLOGY	APPEARANCE OF TONGUE
WHITE TONGUE	The scrapings show yeast organisms, which, however, are not always obtainable	A part or all of the tongue appears dull white as if cauterized with phenol

2. Odors arising from diseases of the soft structures of the oral cavity. All suppurative processes are productive of foul odors. Decomposition or ulceration of tissue in syphilis or malignant tumors causes marked foul odors.

3. Odors arising from diseases of the nasopharyngeal region. Pharyngitis, various types of tonsillar infections, and adenoids are common causes of halitosis. Diphtheria produces a readily distinguishable penetrating odor. Ozena causes one of the most potently vile odors, arising from the nasopharynx in cases of syphilitic ulcers, chronic rhinitis, and nasal tumors.

4. Odors arising from the digestive tract. Certain diseases of the esophagus or the late stages of esophageal carcinoma are productive of pronounced foul odors.

5. Odors arising from bronchopulmonary diseases. Chronic fetid bronchitis is marked by an intense foul odor, and gangrene of the lung with pulmonary tuberculosis by the odor of decayed meat.

6. Odors arising from abnormal metabolic processes. The lung is one of the organs of elimination of toxic products absorbed from the gastrointestinal tract or other diseased part of the body. In marked chronic constipation there is a marked fecal odor of the breath. In diabetes mellitus the fruity odor of the breath (acetone breath) is diagnostic. Scurvy, leucemia, purpura, and similar diseases of the blood are always accompanied by pronounced fetid odors. In chlorosis, a peculiar skatolelike odor of the breath is noted. During menstruation an odor resembling onions may be observed. In cirrhosis, there is a halitosis resembling the smell of decomposed blood. In febrile diseases such as scarlet fever, measles, chicken pox, smallpox, typhoid fever, diphtheria, and influenza there is a temporary but pronounced oral fetor. In yellow fever a peculiar odor is noticed in the expired air. In uremia an ammoniacal odor is noted, this becoming definitely urinous in advanced cases.

7. Odors arising from the presence of absorbed drugs and poisons. Among the absorbed drugs and poisons producing definite odors in the expired air are the arsenical and bismuth compounds. Ether causes a distinct breath odor. Hydrocyanic acid compounds cause a bitter almond odor, and oil of turpentine produces a violet-like odor. Chronic lead poisoning causes a peculiar sweetish metallic odor called the lead breath. A definite breath odor is associated with phenol, lysol, or laudanum poisoning.

8. Odors arising from foods, condiments, and stimulants. Cheese, cooked eggs, and protein substances, when left about the teeth, are ready sources of hydrogen sulfide. Onions and garlic give a definite odor. Yeast produces a beer odor. The use of tobacco causes a halitosis, and opium produces a musty breath odor. In alcoholisum the stimulants impregnate the breath.

The important facts of the anatomy of the tongue and a consideration of the definition and etiology of breath odors have been presented. In Tables I, II,

Table III

General Diseases Affecting the Tongue and Breath

CONDITION	ETIOLOGY	APPEARANCE OF TONGUE
ACROMEGALY	Increased activity of the anterior lobe of the pituitary body	The tongue is thick and prominent and enlarged
ADDISON'S DISEASE	A progressive syndrome due to a disease of the suprarenal gland	Dark spots or streaks may appear on the tongue. These later become deep brown or brownish black
ANGIONEUROTIC EDEMA	Disturbances of the innervation of the blood vessels	The tongue is enlarged, light red in color, with prominent papillae
APOPLEXY	Cerebral hemorrhage and thrombosis	Half of the tongue may be paralyzed, resulting in indistinct enunciation
ARIBOFLAVINOSIS	Riboflavin deficiency	The tongue is purplish red in color and granular. There are deep fissures or desquamated areas resembling geographic tongue. The tongue is painful and there is a burning sensation
BLOOD DYSCRASIAS	The etiology of many blood dyscrasias is not known. Among the dyscrasias are hemophilia, polycythemia, thrombocytopenic purpura, the leucemias, agranulocytopenia, Hodgkin's disease, and the anemias	In polycythemia the tongue shows a bluish-red color. In thrombocytopenic purpura the outstanding symptoms are purpuric spots which are pin-point petechiae with larger areas of ecchymosis. These spots are red at first but later become purplish-red. In myeloid leucemia the tongue becomes congested; in lymphatic leucemia a black discoloration of the tongue due to submucosal hemorrhages is observed; in monocytic and aleucemic leucemia the lowering of gingival tissue resistance renders the tissues susceptible to infection. Agranulocytopenia resembles Vincent's infection. Here the tongue is red and swollen, and there is pain and difficulty in swallowing. In hypochromic and secondary anemia the papillae of the tongue atrophy. In pernicious anemia, the tongue shows changes described as Hunter's glossitis. There is a superficial edema, smooth glossy appearance, pallor, and pain. In most of the cases of the blood dyscrasias there is a fetid odor of the breath
BROMISM	Use of bromine	There is tremor of the tongue and salivation
CHICKEN POX AND SMALLPOX	Acute infectious diseases	The tongue is enlarged
CHRONIC ALCOHOLISM	Great amounts of alcoholic beverages prevent absorption of vital substances	The tongue is swollen, bright red, and bears impressions of the teeth at the sides and tip. In some cases the tongue may be semianesthetic, in others hypersensitive. Some tongues show ulcers along the sides and the tip, and are tremulous
CIRRHOSIS OF THE LIVER	Exact cause unknown. Certain toxins may be responsible	The tongue is coated and the breath smells like decomposed blood
CRETINISM	Congenital deficiency of thyroid substance	The tongue becomes enlarged and elongated until it protrudes from the mouth. The exposed part becomes desiccated and cracked
CYANOSIS	Congenital heart disease	The tongue is blue in color
DELIRIUM TREMENS	Abuse of alcoholic stimulants	The tongue exhibits a muscular tremor when protruded. It is usually covered with a thick white fur
DIABETES	A rise in blood sugar	There is a burning and dryness of the tongue. The breath has a fruity (acetone breath) odor

(*Table continued on next page.*)

TABLE III—CONT'D

CONDITION	ETIOLOGY	APPEARANCE OF TONGUE
DUODENAL ULCER	Functional disturbance of the nervous system. Infection is an important factor	The tongue is coated and the breath offensive
DYSENTERY	*Endamoeba histolytica* and *Shigella dysenteriae*	The tongue is swollen
EPILEPSY	Chronic functional disease	There is an acute diffuse glossitis and the tongue is usually involved in convulsions and may be bitten
ERYSIPELAS	Streptococcic infection	A stomatitis is produced resulting in edema
FOOT AND MOUTH DISEASE	Infection by a filtrable virus acquired by contact with diseased animals	Small yellow vesicles are formed on the tongue. They contain a milky fluid, and are surrounded by dark red, slightly raised zones. The breath is offensive
GASTROINTESTINAL DISTURBANCES: CONSTIPATION, CHRONIC GASTRITIS, ACUTE GASTRITIS, ENTEROCOLITIS	Diseases and affections of the gastrointestinal system	The tongue is coated and may be dotted or stippled. It is yellowish gray in color and there is a bad taste in the mouth. The breath has an offensive odor. In very bad cases the tongue may be a crusted brown, and there may be a burning sensation in it
HERPES	Intestinal disorders of acute infections	There are vesicles on the tongue and there is an accompanying burning and itching sensation
HYPERESTHESIA	Neurosis of the trigeminal nerve	There is an associated pain
HYSTERIA	Psychoneurosis	There is a complete diminution of sensibility and a burning sensation of the tongue
INFANTILE MYXEDEMA	Thyroid hormone deficiency	The tongue is large, fissured, and indented, thus making for clumsy movements
INFECTION AND INJURY OF THE BRAIN	Trauma and diseases involving the brain	There is a tendency toward anesthesia of the tongue
JAUNDICE	Presence of bile pigments in the blood due to duodenal catarrh, liver diseases, etc.	The tongue is yellow along is margins
KIDNEY DISEASES	Disturbances of, and affecting, the kidney	The tongue is enlarged, light red in color, with prominent papillae
MENOPAUSE	Cessation of menstruation	There is a burning sensation of the tongue
METALLIC POISONING: MERCURY, LEAD, BISMUTH, ETC.	Poisoning or overexposure to metals	There is pigmentation of the dorsum of the tongue by the particular metal. The tongue is swollen and red. There is an intense fetor of the breath except in bismuth poisoning, where it is absent
MOELLER'S GLOSSITIS	Allergy, drug eruptions, neural disturbances, nutritional deficiencies, etc.	There are atrophic areas present. These are irregular and limited to the sides of the tongue but may spread over the dorsum. The tongue is bright red and painful
MONGOLIAN IDIOCY	Congenital	The tongue is large and coarsely papillated. It protrudes from the mouth
PARESIS	Syphilis	There is a tremor of the tongue which appears as an irregular quivering movement passing over the tongue in waves
PATHOLOGY OF THE NOSE AND MIDDLE EAR, AND DISTURBANCES OF THE LINGUAL, GLOSSOPHARYNGEAL, AND CHORDA TYMPANI NERVES	Central or peripheral lesions	There may be a diminution (hypogeusia) or complete loss (ageusia) of taste. Involvements of the lingual nerve and chorda tympani affect the anterior and margins of the tongue, and glossopharyngeal involvements affect the posterior

Table III—Cont'd

CONDITION	ETIOLOGY	APPEARANCE OF TONGUE
PELLAGRA	Deficiency in nicotinic acid	There is swelling and redness of the tip and lateral margins of the tongue. The papillae are engorged, then atrophy. The redness is of a fiery, scarlet color due to the shedding of epithelium. When the tongue becomes denuded, it has a bald beefy appearance (bald tongue of Sandwith). The tongue is frequently fissured. There is an associated tenderness and pain
PEMPHIGUS	Unknown	The condition produces a stomatitis which results in an edema of the tongue
PLUMMER-VINSON SYNDROME	The condition is seen in women past middle age who are edentulous and thin	The tongue is shrunken and smooth and there is difficulty in swallowing
PNEUMONIA	Infection by pneumococci	The high fever produced by this infection causes the tongue to appear enlarged, furred and coated, with an accompanying oral fetor
RHEUMATIC FEVER	Infectious diseases resulting in heart disease	The tongue is enlarged and coated
SCARLET FEVER	Infection by the beta hemolytic streptococcus. The portal of entry is usually via the nasopharynx	The strawberry or raspberry tongue is diagnostic. There is a desquamation of the surface epithelium leaving a red surface with enlarged and inflamed papillae. The enlarged fusiform papillae become red and prominent and project over the white desquamations of the filiform papillae. This is the strawberry tongue. When the desquamation of the filiform papillae is complete, the granular purplish-red appearance of the mucosa of the tongue gives rise to the raspberry tongue. The breath is very offensive
SCURVY	Vitamin deficiency	The tongue is enlarged. The primary symptoms are in the gums and mucous membranes. There is a pronounced fetid odor of the breath
SHOCK	Severe hemorrhage	The tongue is dry, indicating dehydration
SYPHILIS	Infection by spirochete of syphilis	Secondary lesions appear on the tongue in the form of mucous patches usually multiple. The most frequent type is round with a slightly rough surface, glistening whitish-gray in color, and slightly raised above the surface. When scraped off, a raw bleeding surface is exposed. The tongue may contain large numbers of spirochetes with a resulting endarteritis. Later there occurs secondary atrophy of the papillae, which leaves the dorsum bare. Irritation of this produces an epithelial hyperkeratosis called glass tongue. In tertiary syphilis, gumma or nodular masses form and enlarge slowly and painlessly, until the whole tongue is involved. There is a tendency to ulcerate. In syphilitic meningitis there is an inability to protrude the tongue
TYPHOID FEVER	Infection by the typhoid bacillus	The tongue is heavily coated with grayish fur, with a red border at the sides. There is an offensive odor of the breath
TYPHUS FEVER	This disease is favored by filth. The causative organism is transmitted by lice	There is a blackness and dryness of the tongue. The breath has a peculiar musty odor

and III an attempt has been made to list some of the pathologic conditions that affect the tongue alone and the tongue and breath together. There have been many conditions that have not been included because of their rarity and relative lack of interest to the dentist.

CONCLUSIONS

The preceding tables are an attempt to present to the reader the importance of the tongue and breath in diagnosis. The list of diseases presented is by no means complete as only the commonest pathologic conditions have been chosen. In the opinion of the writer, the subject is one that could be adequately covered only in a textbook.

It cannot be too greatly stressed that the dentist will have to prove his ability to cope with disease affecting the mouth and its associated parts, if dentistry is to be considered an important branch of medical science. The appearance of the tongue and the odor of the breath offer ideal opportunities for the dentist to prove himself an oral specialist, and, in addition, to be a definite asset to the physician in the early detection of systemic disease. If this opportunity is not immediately seized, then the future of dentistry as one of the important and necessary professions will be questionable.

SUMMARY

The dentist should diagnose diseases of the mouth and its contents, and should be well trained in recognizing systemic diseases by the appearance of the tongue and the odor of the breath. These have been presented under the following headings:

1. Congenital defects and deformities of the tongue.
2. Local diseases affecting the tongue and breath, and diseases of the tongue itself.
3. General conditions affecting the tongue and breath.

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HORIZONTAL PIN FIXATION FOR FRACTURES OF MANDIBLE USING PIN GUIDE

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MANY professional articles as well as those for the layman have been written concerning fracture appliances which permit function during healing. It is a recognized fact that normal physiologic function must be limited to some degree. However, any use that can be made of a reduced fracture during treatment is a distinct advantage.

There have been many methods of treating fractures of the mandible, all having advantages and disadvantages. Interdental immobilization by wiring is a simple method, quickly applied, but does not permit mandibular motion or facilitate good oral hygiene. The use of intraoral splints permits mandibular motion but prevents proper cleansing of the area involved. Extraoral appliances (Stader splint) is not the answer to most civilian fractures because cleansing and caring for the face is very difficult under this appliance. There are many advantages in its use, however, particularly in cases in which there has been a loss of bone (war wounds), and it is desired to maintain space, limiting the deformity and simplifying the reconstructive surgery.

It was in an effort to find some means of fracture fixation that would permit mandibular motion and good hygiene that the horizontal pin method was devised. This procedure consists of one or two pins (Kirschner wire, large) drilled horizontally through the body of the mandible, across the fracture with both ends of the pin imbedded in the cortical bone. The wire can be cut short and the skin raised over the end, leaving no external evidence of the fixating pin.

This type of fracture immobilization permits normal physiological mandibular function. The patient is able to continue with his work and carry on a regular routine of life, being limited only to a liquid or soft diet.

After having drilled a few of these pins in the mandible, one is soon convinced of the necessity of some type of a pin guide. I designed and had Jack Pava, brace mechanic at Hoff General Hospital, make the pin guide shown in Fig. 1. The tube *A D* directs the wire to point *B*. This device eliminates the guesswork and hazards of freehand drilling.

The technique used is as follows: A minimum of loop wires is applied to the upper and lower teeth. Intermaxillary rubber bands are used to re-establish proper occlusion and alignment of the fragments. This is a very important step. If normal interdental relation is not correctly established, the resulting malocclusion will produce traumatic pressures with the resulting loosening of the

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pin and possible failure of union. Block anesthesia is used with local infiltration in the region of the symphysis and posterior border of the mandible. The pin guide is applied by first inserting the point *B*, Fig. 1, into the posterior border of the mandible. The point of the guide tube (Fig. 1, *A*) is then positioned into the area lateral to the symphysis. The wing pin (Fig. 1, *C*) is then tightened as the pin guide is rotated on its axis (Fig. 1, *A B*). These points are so beveled that they will drill themselves into the cortical bone. When the pin guide is firmly clamped into position, posteroanterior and lateral jaw x-ray films are taken. This permits checking the position of the director before the pin is drilled into the mandible. The pin is then screwed out of the tube at Fig. 1, *D*, and the large Kirschner wire is run through the directing tube and drilled into position. It is necessary to disassemble the two halves of the clamp by releasing the wing pin to remove it from its position. Digital examination is then made to find the posterior end of the pin. If it cannot be located, the drilling is continued until the end can be felt just through the posterior border of the ramus. The pin is cut short enough to permit pulling of the skin and subcutaneous tissue over it. A small dressing is applied over the anterior and posterior pin holes in the skin and removed in two or three days. The mandible is subsequently roentgenographed to check the pin position, and then the interdental wires and rubber bands are removed. If it is felt that additional support is necessary to prevent rotation of the fragments, a figure-of-eight wire can be applied to the teeth on either side of the fracture.

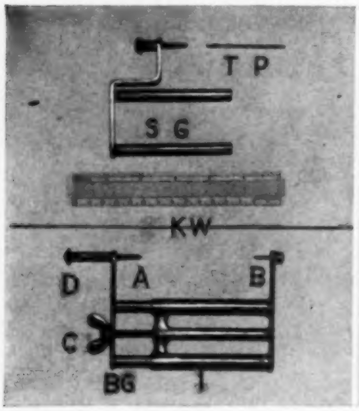


Fig. 1.


Fig. 2.


Fig. 3.

Fig. 1.—*A* and *B*, Tribeveled points; *D*, thumb nut and pin which when removed opens tube *D-A*; *C*, wing bolt for clamp adjustment; *B-G* and *1*, mandibular body guide (see Fig. 2); *S-G* substituted for *B-G* with *1* makes symphysis guide (see Fig. 3); *K W*, Kirschner wire (large); *T-P*, threaded pin (stainless steel) 0.081 by 58 mm.

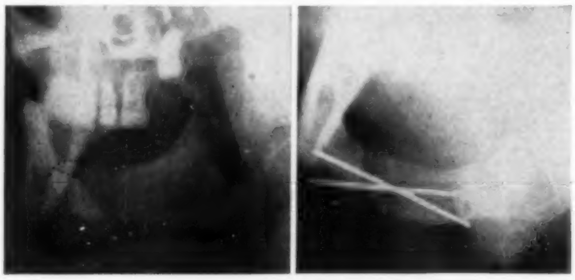
Fig. 2.—The body guide applied to the mandible (inferior view).

Fig. 3.—Symphysis guide applied to mandible (inferior view).

The pin can be removed in about six weeks under local anesthesia. The end of the pin is located by digital examination, and about 1 c.c. of 2 per cent procaine is infiltrated into the area. The operator makes a small stab incision, feeling the end of the wire with the scalpel. The surrounding tissue is compressed until the end of the pin extends through the incision. It is grasped in a heavy plier and withdrawn by a rotating motion.

It has been rather surprising how firmly the pins have been held in position, and at times they require considerable turning to free them.

A series of twelve completed cases have been treated in this manner at Hoff General Hospital, U. S. Army. We have had no infections, and all have healed with excellent results. The patients have been very comfortable during treatment, and a casual observer would not know they were being treated for mandibular fractures. In fact, I have had two patients ask for this form of treatment. They were patients with interdental wiring applied at their station hospitals, then transferred to our hospital, and were able to observe this type of fixation on other patients in their ward. When patients ask for it, the factor of comfort must be of some importance.



Figs. 4 and 5.—Case 1.

Case 1.—C. A., aged 29 years.

Diagnosis: fracture of the mandible, ununited, oblique, region of the right symphysis, with overriding edentulous fragments. Injury sustained in automobile accident thirteen years prior to entrance into military service.

General anesthesia: nitrous oxide oxygen and ether was used and open reduction was carried out. An incision was made along the inferior border of the mandible over the fracture site, and the periosteum was reflected from the fragments. The ends (two) were freshened by means of an electrically-driven circular saw. The fragments were then approximated and held in position by two crossed horizontal pins (threaded 0.081 stainless steel). The periosteum and subcutaneous tissue were approximated with catgut; the skin was sutured with 5.0 silkworm gut (Figs. 4 and 5).

This patient had an uneventful recovery. The pins were removed eight weeks after operation under local anesthesia. The fragments were healed in excellent position, and the patient was returned to full duty.

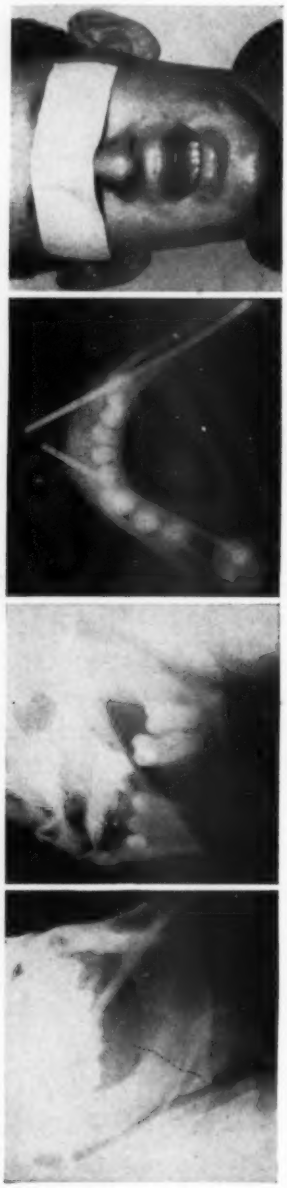
Case 2.—K. E. J., aged 29 years.

Diagnosis: fracture of mandible, compound, comminuted, complete, bilateral, regions of left first molar and right third molar, accidentally incurred during supervised athletics.

The anesthesia was produced by mandibular block, with local infiltration. A minimum of looped wires was applied to the teeth, and intermaxillary rubber bands were used to re-establish the occlusion and alignment of the fragments. The pin guide was applied and adjusted into position. A large Kirschner wire was drilled horizontally through the body of the right and left mandibles. A

figure-of-eight wiring was applied to the teeth, left first and second bicuspids and first molar, to aid in preventing rotation of the fragments (Figs. 6, 7, 8 and 9).

This patient had an uneventful recovery. The pins were removed in six weeks, and the soldier returned to full duty.



Figs. 6, 7, 8, and 9.—Case 2.

Case 3.—P. S. I., aged 22 years.

Diagnosis: fracture of the mandible, compound, comminuted, complete, unilateral, region of the left second molar. The fracture was sustained during a fight with another soldier.

Mandibular block, with local infiltration anesthesia was used. Occlusion was re-established and the fragments were realigned by the use of the loop

method, with intermaxillary rubber bands. A Kirschner wire was drilled from the posterior border of the ramus anteriorally, making its exit on the lateral surface of the mandible in the region of the mental foramen. The wire was directed in this position by inserting point *A*, Fig. 1, of the pin guide, into the posterior border of the ramus, and point *B*, Fig. 1, held into the lateral surface, just above the inferior border, in the region of the mental foramen. This mandible was oval in shape, the patient's face being very round, making it impossible to drill the wire from the symphysis to the posterior border of the ramus (Figs. 10, 11, and 12).

There were no complications, and the pin was removed six weeks after operation, under local anesthetic. He was returned to full duty the following week.

Figs. 10, 11, and 12.—Case 3.

Figs. 13, 14, and 15.—Case 4.

Case 4.—A. T. S., aged 19 years.

Diagnosis: fracture of the mandible, compound, comminuted, complete, unilateral, region of the left first molar. Patient was injured when he was struck on the jaw by a civilian. The anesthetic used was mandibular block, with local infiltration. Occlusion was reestablished by the loop method and intermaxillary rubber bands. The pin guide was applied, and a Kirschner wire (large) was drilled horizontally through the body of the mandible, extending from the left symphysis to the posterior border of the ramus (Figs. 13, 14, and 15).

The pin was removed in six weeks, and the soldier was returned to duty four days later. There were no complications during treatment of this case.

This method of treating fractures of the mandible was thought to be new and was so demonstrated at the Oct. 19, 1942, staff meeting of the Hoff General Hospital, U. S. Army. However, articles were later found dating the treatment as early as July 9, 1932,[2] and Sobye[4] published a series of twenty-five cases

in 1939. This treatment was also reported by Meade[3] in 1935 and Brown[1] in 1942.

These articles have all reported a tendency of the pin to loosen after operation. This has not been the experience at this hospital. The pins removed six weeks after operation seem to offer about the same resistance as those which were removed for redrilling. If the pin is confined to the intermedullary bone, the loosening is negligible. The pictures demonstrated in the articles by Ipsen, Meade, and Sobye give one the impression that the pins have been drilled from the right to the left mandibles, across the floor of the mouth. It is possible that the muscle traction in the floor of the mouth accounts for the reported loosening of the pin.

The question might well be asked: What will happen to the mandibular nerve if the pin is drilled into the canal? An effort is made to place the pin below the canal, but if the Kirschner wire should enter it, I believe it to be of no importance. The wire is smaller in diameter than the canal, and as the pin passes through the canal, the nerve will be pushed aside, causing little or no damage.

It is very difficult to judge the trauma associated with drilling the pin in the canal because of the anesthesia usually present with mandibular fractures. In the series of patients treated at this hospital, it has been impossible to observe any change in the degree of anesthesia. Four of the postoperative x-ray films have shown the pin and mandibular canal in contact. However, one must keep in mind that the pin could be placed in a position medial or lateral to the canal, and the shadows superimposed. Therefore, the pin may or may not have been drilled into the canal.

CONCLUSION

Positioning the horizontal pin or Kirschner wire (large) through the mandible requires accurate drilling. The pin guide will direct the wire into only one position, points *A* and *B*, Fig. 1. Therefore, the success in positioning the pin correctly depends upon the proper adjustment and alignment of the guide.

ADVANTAGES

The comfort of the patients is a paramount factor. They are able to keep their mouths clean, to eat without the laborious effort of sucking everything through their teeth, and to speak distinctly. In other words, the patient has normal physiologic mandibular motion, being limited only to a liquid or soft diet. There is no external appliance to interfere with the cleaning of the face and to detract from the appearance of the patient.

The only disadvantages of this method, compared to intraoral immobilization, is that this procedure must be done aseptically and requires an operating room to carry out the technique.

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CASE REPORT OF HEMANGIOMAS OF TONGUE AND CHEEK TREATED WITH RADIUM

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MISS D. S., white, aged 35 years, applied for treatment of hemangiomas of the tongue and right cheek upon the advice of her dentist, and the suggestion of a friend who had a similar condition treated at our institution successfully. She had no other complaint.

The patient wore glasses for the relief of headaches. She had had an appendectomy fourteen years before; otherwise, her medical history was negative.

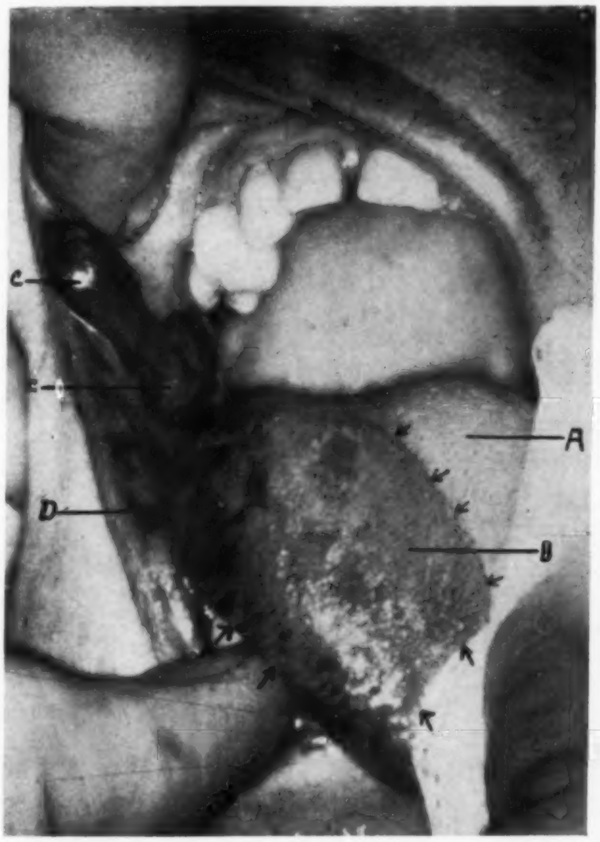

Fig. 1.—*A*, Normal half of tongue; *B*, hemangioma on tongue; *C*, hemangioma in cheek; *D*, hemangioma in cheek.

Physical examination was essentially negative. Blood pressure was 140/90. Urinalysis and Kline tests were negative. There were no hemangiomas aside from those in the oral cavity, nor were any other abnormalities present. She had most of her teeth, although the lower right first and second molars were missing. Her mouth was in a fairly good state.

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Examination disclosed the presence of a large hemangioma of the tongue, occupying the anterior two-thirds of the dorsum on the right side (about 7 cm.). Another was present on the inner surface of the right cheek, about 5 by 2.5 cm., extending forward to involve the vermilion border of the lower lip. Both were bulky (Fig. 1). In addition, there were a 1 cm. hemangioma of the ventral surface of the left side of the tongue and an 0.5 cm. hemangioma on the vermilion border of the lower left lip.

All were present since infancy and had never caused the patient any difficulty. Only the two large tumors on the right side were to be treated at the time.

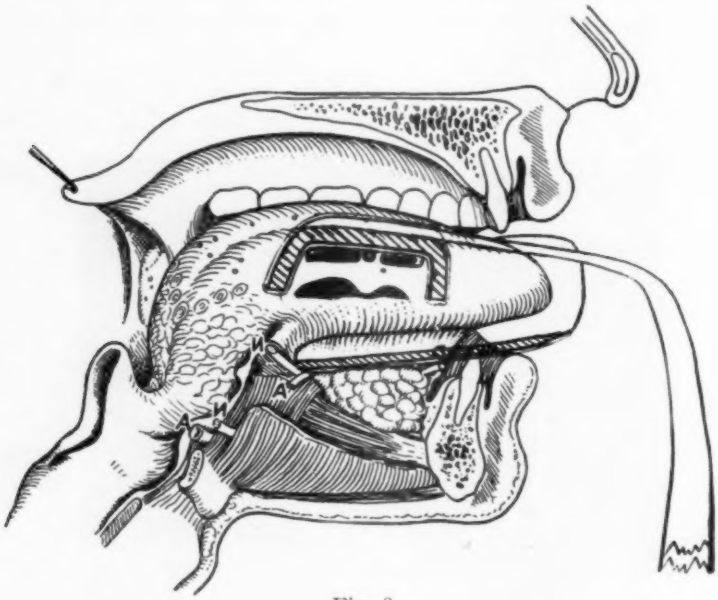

Fig. 2.

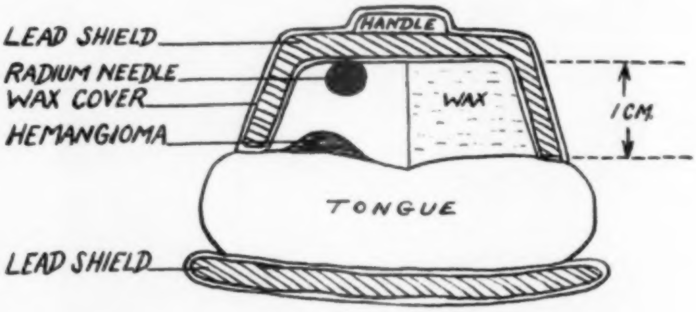

Fig. 3.

Therapy consisted of the application of radium encased in small platinum tubes about 2 mm. in diameter and 1 cm. in length. Appliances were constructed in such a way as to serve both as a medium for holding the radium in proper position in relation to the hemangiomas and to protect the neighboring normal tissues from scatter radiation. Prior to the construction of this device, a molar (lower right) and the lower right first and second bicuspids, in the line of radiation and not essential to future prosthetic restoration, were extracted.

A description of the construction of the appliances follows: The first step was to cut a U-shaped piece of sheet lead, $\frac{3}{16}$ inch in thickness, in such a manner that it fit underneath the tongue when the latter was fully extended, and still left a border of $\frac{1}{2}$ inch. This form was then put into position, with the tongue extended, and a plaster impression was taken of the tongue. A stone model was poured. Using the latter as a guide, another lead sheet, $\frac{1}{8}$ inch thick, was shaped so as to cover the area of the lesion and yet leave enough space for the wax to hold the radium capsules and also to allow for a distance of 1 cm. between the radium and the tissue.

This form was then attached to a metal tongue depressor instrument. Wax, in thickness about 1 cm., was added to the surface for holding the radium. Next, all exposed lead was covered with baseplate wax to prevent secondary irradiation. Radium capsules were put in place; the lower lead sheet, also wax-covered, was put beneath the tongue; the upper part was placed. The patient was now able to hold the appliance in position for the prescribed length of time (Figs. 2 and 3).

The device used in the cheek was made in the following manner: A piece of wooden tongue depressor blade was used as a tray around which was placed a bulk of soft modeling compound. This was then put into the mouth on the side of the lesion, and the patient was told to bite into the compound. The area pushing out the cheek was molded by gently massaging the cheek. The patient was instructed to mold the lingual portion with the tongue.

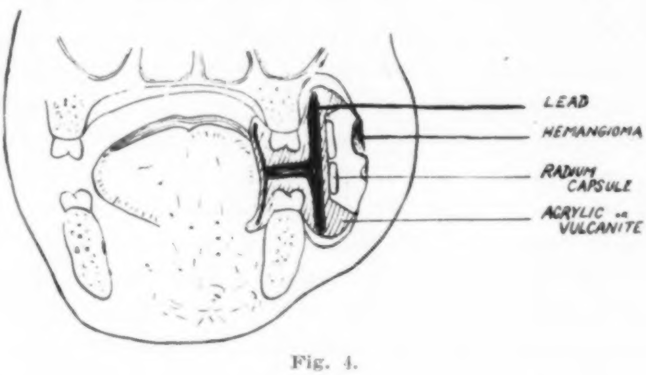

Fig. 4.

After the compound hardened, it was removed, the window cut out, flasked, boiled out, and then the mold tinfoiled. Some clear acrylic was put into the mold to partly fill it, and lead ($\frac{1}{8}$ inch thick) was placed in position as shown in Fig. 4.

The mold was then filled with the acrylic and processed. The appliance was polished to prevent irritation to the tissues. Radium capsules were placed in wax put in the window. Retention of the appliance is maintained by the teeth biting into the spaces produced by them originally in the impression compound.

The radium succeeded in reducing the bulk of both hemangiomas treated. On the tongue, only the posterior third of the hemangioma remains. On the cheek, however, the whole lesion is still present, though greatly reduced in bulk. The patient is now receiving injections with a sclerosing solution in a further attempt to reduce the size of the hemangioma in the cheek.

THE REPAIR OF BONY AND CONTOUR DEFORMITIES OF THE FACE

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Displacement or loss of supporting structure of various parts of the face as a result of faulty development, injury, disease, or operation is accompanied by impairment of function and appearance. The principal facial deformities of developmental origin are cleft palate, unshapely contour of the external nose, and malrelationship of the upper and lower jaws resulting in undue prominence or recession of the chin, open bite, and other disturbances of occlusion of the teeth. Acquired deformities involve displacement or loss of supporting tissues (bone and cartilage), as well as overlying soft tissues, of varying extent, causing cosmetic or functional disturbance or both.

The means at our disposal for the replacement of lost or displaced supporting tissues following injury, disease, or operation are the use of soft tissue flaps, cartilage, bone, fascia lata, dermal grafts, and artificial appliances. Some bony defects accompanied by soft tissue loss can be satisfactorily repaired with soft tissue flaps alone when the bone loss involves an immovable part such as the hard palate or the outer surface of the maxilla. In the smaller palatal defects, sliding or pedicle flaps of oral or nasal mucous membrane may be used, while for the larger ones the introduction of pedicled flaps of skin may be necessary. Where a large bony defect is accompanied by loss of skin or mucous membrane on both sides, both epithelial surfaces should be replaced. Artificial substitutes are sometimes necessary to replace losses of bone and soft tissues of the upper jaw, being more satisfactory than surgical restorations under some circumstances. In some cases of palatal defect, for example, it may be possible to close the opening by surgical means, but the tissues thus repaired do not afford satisfactory attachment for artificial dentures. Artificial substitutes are occasionally preferable in external defects about the face where the extent of the tissue loss or the age of the patient militates against satisfactory surgical repair.

For the deeper and more extensive contour defects of the frontal region, nose, cheek, chin, or other parts of the face, costal cartilage forms the most satisfactory replacing substance. Several foreign materials (such as celluloid and ivory) have from time to time been advocated for this purpose. They have an advantage in that they are easily inserted without mutilating other parts of the body, but they are subject to all the disadvantages of any foreign body; for example, extrusion is possible at any time. At one time paraffin was extensively used, but it should have no place in plastic surgery of the face, as it is difficult to control in injection, sometimes causing unsightly disfigurements, undergoes changes in shape, and may cause embolism and tumor formation. In late years, vitallium and, more recently, tantalum have been used with encouraging results. Transplanted bone as a support for the bridge of the nose is employed successfully by some surgeons. We prefer costal cartilage for this purpose, because it

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rarely undergoes absorption when imbedded in soft tissues and is not so susceptible to infection as bone. Costal cartilage is not difficult to obtain in almost any quantity desired and is easily trimmed to suitable size and shape. While living autogenous costal cartilage is undoubtedly preferable to any other, in recent years many operators have successfully used preserved cartilage from other individuals and even from cadavers. The use of preserved cartilage has the advantage in that unlimited quantities are available, and the patient does not have the inconvenience of the chest operation added to that on the face. Living autogenous cartilage, on the other hand, is less likely to undergo changes after transplantation. Costal cartilage has a tendency to change shape by curling, resulting in an unsightly deformity. To overcome this, Gordon New exposes the cartilage after removal to live steam for a minute or two. This causes the maximum amount of curl to take place immediately, after which the cartilage can be trimmed to proper size and shape and no further changes will occur. Of course, the steaming destroys the vitality of the cartilage. Costal cartilage does not form a firm union with bone; hence, it cannot effect a stable restoration of continuity in a movable bone such as the mandible. For filling gaps to restore continuity of the mandible, bone transplants alone are satisfactory.

Displacement of bony structure alone, or combined with loss of substance, may be responsible for facial deformity. In some cases of this type, correction of the deformity may be attained by mobilization of the displaced portion of bone and replacement in normal position. This is not always possible, especially where the displacement is of long standing, such as in the case of an old depressed fracture of the nasal or malar bones. Here, correction is more satisfactorily brought about by building out the contour with added bone or cartilage at the site of the depression.

After a fracture of the mandible, with or without loss of substance, union may take place with shortening at the site of fracture, resulting in facial asymmetry and functional disturbance. The same sort of deformity may follow loss of a portion of the mandible from necrosis or operation for removal of a neoplasm. Here, the displacement first must be corrected by osteotomy at the site of malunion or division of adhesions holding displaced fragments in a contracted position, and by reduction of fragments to a position as normal as possible, followed later by bone grafting if a gap remains that is too wide to be bridged by spontaneous bone regeneration.

Osteotomy of the mandible may be carried out with a narrow chisel at the site of malunion but is usually done with less trauma by means of a Gigli saw. A small incision through the skin, not more than one-half inch long, is made to the lower border of the mandible at the point of malunion. A full-curved pedicle needle is passed through the incision, close to the inner surface of the bone, until the mucous membrane of the mouth is pierced. By means of a wire through the eye of the needle, a Gigli saw is carried on the inner surface of the bone with one end in the mouth and the other emerging through the skin incision. The mandible is then sectioned with the saw from the inner side out through the fracture line. The skin incision is sutured and usually heals by first

intention, leaving practically no visible scar. The bone fragments are now reduced and treatment is as for a recent fracture, with or without loss of substance. The method of fixation of the fragments to be used depends upon the availability of teeth. In most patients with sufficient teeth in each fragment and in the upper jaw, we find that simple wiring methods, such as the eyelet or the arch bar methods, are most easily applied and give adequate fixation. The more elaborate splints are reserved for patients in whom teeth are not adequate for use of the simpler methods.

Bone grafting is sometimes employed for repairing defects of the cranium and contour losses of various parts of the face, but its most frequent use is in restoring continuity and replacing losses of substance of the mandible resulting from trauma, necrosis, or resection for treatment of disease.

Statistics from World War I indicate that about 11 per cent of gunshot fractures result in nonunion and require bone grafting. The nonunion is caused principally by the large loss of bone substance and inability of the repair mechanism to bridge the gap when the collapsed fragments are drawn apart and fixed in proper position. The loss of bone is not so often due to primary destruction as to extensive shattering followed by infection and necrosis. In comminuted fractures, proper early fixation and conservation of viable bone fragments will do much to render bone grafting unnecessary later.

The object of treatment in patients with loss of substance of the mandible from whatever cause is primarily restoration of the function of mastication. This is attained by restoring the normal occlusion of the remaining teeth and stabilizing the jaw by filling in the lost continuity of the bone. A secondary objective is the building out of contour of the face for improvement in appearance. In patients with loss of substance it is very rarely desirable to obtain union by bringing the ends of the fragments together, sacrificing the occlusion of the teeth. This results in a crippling of the masticatory function and an increase in the external deformity.

Where bone grafting is contemplated, the preoperative treatment of patients with losses of substance of the mandible is as follows: removal of all septic foci, reduction, and fixation in such a position that the normal occlusion of the remaining teeth is restored. Septic foci include roots of teeth projecting into the area of lost substance, other teeth showing evidence of periapical or periodontal disease, bony sequestra, metallic foreign bodies, and infection in the adjacent soft tissues. No operation to restore the continuity of the bone should be attempted until all sources of infection have been removed and until at least three months have elapsed after all sinuses and septic wounds have healed.

Reduction is brought about in cases of nonunion by manipulation, followed immediately by the application of wires to fix the upper and lower teeth together, or, if the existing teeth are not adequate for this method, then the more elaborate interdental splints are used. Where the bone fragments are not freely movable and cannot be reduced by manipulation before application of measures of fixation, owing to the presence of fibrous adhesions, it becomes necessary to sever these adhesions at a preliminary operation, apply the fixation, and then wait several weeks for healing of the soft tissues to occur before performing the bone-grafting operation. As long as there is any possibility of contamination of the

wound with oral secretions, bone grafting is contraindicated. In nearly all cases of this type the preliminary operation requires cutting through the oral mucosa, which renders absolutely necessary postponement of the bone grafting until complete healing has occurred.

Types and Methods of Bone Grafting in the Mandible.—In many of the patients after World War I restoration of continuity was brought about by a pedicled graft from the mandible itself, the pedicle consisting of attached muscle. This method was first described by Bardenheuer in 1893 and was popularized by Cole, of London. We have not used this method recently because we found that it produces undue distortion of the soft tissues of the floor of the mouth and neck and is not suitable for large losses of bone substance, especially in the region of the angle and ascending ramus of the mandible. We consider also that the cortex of the tibia is not very suitable as a source of bone graft for the mandible because of its extreme density and consequent resistance to penetration of new blood vessels in the process of consolidation. Also, fracture of the tibia after removal of a thick graft is not unknown. Rib grafts have sometimes been used for these defects but are usually too thin. During the past twenty-four years we have limited ourselves to two methods, each having fairly definite indications; namely, the osteoperiosteal graft from the tibia and the graft from the crest of the ilium.

1. *The Osteoperiosteal Method of Delagenière:* The osteoperiosteal graft contains all the elements necessary for osteogenesis, is flexible, and is easily adjustable to the size and shape of the defect. The technique of removal and insertion is simpler than that of any of the other methods. It causes no disability in the leg. It requires longer to obtain complete consolidation than by other methods, and no dependence, of course, can be placed on the rigidity of the graft itself for maintenance of the mandibular fragments until consolidation has occurred. While this form of graft can be used for losses of substance of the mandible of almost any extent and any position, we usually reserve it for defects of 2 cm. or less and for patients in whom the external contour of the face shows little or no deficiency. It is also to be selected for use in children, because the crest of the ilium shows very little ossification before 15 or 16 years of age.

2. *Graft From Crest of Ilium:* A graft from the crest of the ilium was first used in Germany by Lindemann and was the method of choice also of Gillies and his co-workers at the Queen's Hospital at Sidcup, England. This type of graft is preferred if the gap in the mandible exceeds 2 cm., and especially if the external contour of the face shows the deficiency. The crest of the ilium furnishes a large piece of bone of porous structure closely resembling that of the mandible, it is easily penetrated by new vascular supply, and it can readily be cut to suitable shape. The disability produced by removal of the graft is quite temporary and the danger negligible.

The technique of grafting the mandible by either of these methods has been fully described elsewhere, so further space will not be taken up with it here, except to say that exposure and preparation of the mandibular fragments are done before removal of the graft from the donor site in order that the size of the graft required may be determined and also on account of the possibility of open-

ing into the mouth while preparing the bed for the graft, an accident that would render useless the application of the graft at this time. Another reason for this order of procedure is the desirability of protecting the viability of the graft by placing it in its new position as soon as possible after removal from its original site.

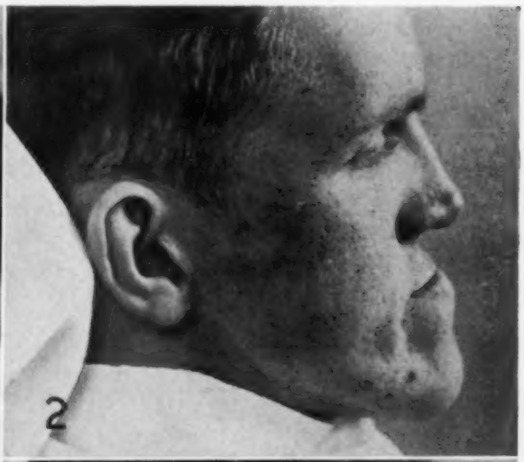
Postoperatively, fixation of the jaws must be maintained until consolidation is well advanced; that is, for at least eight weeks. Regeneration may be checked by x-ray examination at monthly intervals, but the final test of consolidation is by clinical examination after disconnecting the upper and lower teeth.

The following cases illustrate several of the features mentioned in this article:

Case 1.—In Case 1 is illustrated protrusion of the mandible. The patient was first seen at the age of 18 years in consultation regarding a protrusion of the mandible which had gradually become more noticeable with growth. Physical examination revealed nothing having any bearing on this condition. The lower anterior teeth were about three-fourths of an inch in front of the corresponding upper teeth, and only the last molars were in occlusion (Fig. 1). The forward position of the mandible caused a very noticeable prominence of the chin (Fig. 2). There was marked interference with the function of mastication. Impressions were made of the teeth and plaster casts prepared to determine whether a satisfactory dental occlusion could be obtained by setting the mandible back. This being found possible, an operation was decided upon. Preceding the operation, half-round arch bars were applied to the upper and lower teeth to serve for postoperative fixation. Under intratracheal ether anesthesia introduced through the nose, each ascending ramus of the mandible was sectioned horizontally by a Gigli saw introduced through small skin incisions behind and in front of the ramus by means of a modified Blair pedicle needle. This permitted the main part of the mandible to be slid back on these cuts, bringing the lower teeth into good occlusion with the upper. The upper and lower teeth were then fixed in occlusion by connection of the arches on the teeth with tie wires. Immobilization was maintained for six weeks, after which union in the new position was found to be fairly solid. The wire fixation was replaced by intermaxillary elastics for about ten more days, the patient being then discharged. Figs. 3, 4, and 5 illustrate the result of the operation.

Case 2.—In Case 2 is illustrated the use of costal cartilage to restore contour in the frontal region. The patient sustained a crushing injury in an automobile accident with loss of bone in the left frontal region. This left him with a marked depression, the overlying skin being intact except for a vertical scar (Fig. 6). An incision was made through the old scar line and the skin on each side undermined over the depressed area to form a pocket. Into this was inserted a piece of autogenous costal cartilage of suitable size and shape to restore the contour of the forehead. The skin wound was closed with interrupted silk sutures (Fig. 7).

Case 3.—In Case 3 is illustrated a contour defect of the right frontal region with loss of both soft tissues and bone. The patient was thrown from a moving automobile into the road, where he landed on his face, resulting in a compound



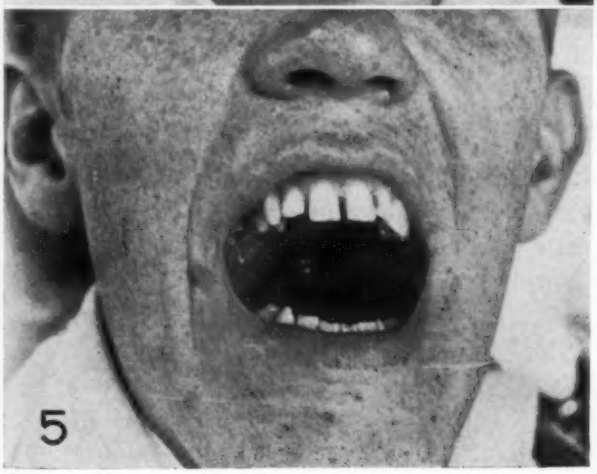


Fig. 1 (Case 1).—Showing advanced position and malocclusion of lower teeth.

Fig. 2 (Case 1).—Undue prominence of chin.

Fig. 3 (Case 1).—Correction of occlusion by bilateral osteotomy through ascending rami and setting back of mandible.

Fig. 4 (Case 1).—Improvement of profile after operation.

Fig. 5 (Case 1).—Showing ability to open mouth after operation.

comminuted fracture of the right side of the frontal bone with avulsion and laceration of overlying soft tissues. Fig. 8 shows the condition of the patient several months later when the defect was ready for definitive repair. On the right side of the forehead there was a broad deep defect, involving the bone in part, covered with healed scar. One small area had been covered with unsightly pinch grafts, no doubt to hasten healing. There was a small opening into the frontal sinus. The extent of the area precluded repair by sliding flaps from the surrounding frontal region. The depth of the defect and the opening into the frontal sinus rendered a free skin graft unsuitable. Consequently, a tube pedicle flap from the right clavicular region was transferred to the forehead in several stages at three-week intervals. The procedure is shown in Figs. 9, 10, and 11. Fig. 12 shows the final stage of the flap transfer. Later, the eyebrow was restored by a free full-thickness graft from the scalp.

Fig. 6 (Case 2).—Marked depression left frontal region due to loss of bone.
Fig. 7 (Case 2).—Elimination of depression by implantation of costal cartilage.

CASE 4.—Brown hairy congenital nevus of face had existed since birth in the patient in this case, and the boy was presented for treatment at 6 years of age. The lesion was dark brown in color and slightly elevated; the surface was rough and covered for the most part with a heavy growth of furlike hair. It involved the entire left cheek, extending into the temporal region, the entire lower eyelid and part of the upper eyelid, the left side of the nose, including the ala, and passed over on to the right side of the nose (Fig. 13). These growths are not radio sensitive, and surgical excision in this case offered the only possibility of successful treatment. Three ways of repairing the defect were considered: (1) Multiple partial excision. In growths and scars involving a moderate area in the cheek, this method is excellent in that it gradually advances the surrounding healthy skin and succeeds in covering the area with skin of texture and color normal for the part, with a very small amount of scarring. In the present case,

Fig. 8 (Case 3).—Healed deformity of right frontal region. Loss of bone and soft tissue with opening into frontal sinus.

Fig. 9 (Case 3).—Stage in transfer of tube pedicle flap from clavicular region for repair of forehead defect.

Fig. 10 (Case 3).—Another stage of transfer of flap to forehead.

Fig. 11 (Case 3).—Another stage of transfer of flap.

Fig. 12 (Case 3).—Flap spread out to cover forhead defect.

the extent of the area involved precluded the use of this method. (2) Complete excision, followed by immediate application of a split-skin graft. This was also rejected, partly because of the inability of a free graft to furnish sufficient contour, and also the appearance of the grafted area would leave too much to be desired. (3) Use of a pedicled flap. This method was finally selected, because it supplied sufficient contour, with a better chance of complete take of the transplanted tissue. It was realized that there would be difficulty in matching the rest of the face in color and texture. At the first operation a tube pedicle was prepared on the left side of the abdomen from a strip of skin and subcutaneous tissue three inches wide and eight inches long. Three weeks later the lower end of the tube was cut loose and sutured into a raw area exposed by turning a flap on the radial side of the right wrist (Fig. 14). Restriction of movement during healing was adequately managed by means of adhesive plaster strips. The next step, three weeks later, consisted in disconnecting the upper end of the tube from the abdomen and carrying it up on the wrist to the face where it was sutured into a raw area in the left temporal region created by excision of part of the nevus. Sufficient fixation for good healing was obtained by holding the arm in the Velpeau position with adhesive plaster and a bandage. Plaster of Paris was unnecessary (Fig. 15). After a further three-week period the flap was disconnected from the wrist and the tubed portion opened out and spread to cover the raw area on the face after excision of the major portion of the growth. Two or three further operations were necessary to smooth edges here and there, and a full-thickness preauricular skin graft was applied to the left lower eyelid. Although there is some discrepancy in color and texture of the skin of the flap as compared to the rest of the face, it is hoped that with time this will become less noticeable (Fig. 16).

Case 5.—In this case is illustrated the deformity of contour of the face and interference with function due to malunion in a fracture of the mandible. The patient was edentulous and sustained a fracture of the left ascending ramus of the mandible. Owing to the absence of teeth, no reduction or fixation was applied after the injury, with the result that his fracture united in malposition, the left side being shortened. This caused the chin to deviate to the left. The mouth opening was greatly restricted, and this and the malrelation of the upper and lower alveolar arches prevented the wearing of artificial teeth (Figs. 17 and 18). Correction was carried out in the following way: An osteotomy was performed at the site of the fracture in the left ramus of the mandible. This permitted the main portion of the lower jaw to be carried to the right and the chin to the midline position. Fixation by means of the teeth being impossible, a circumferential brass wire was passed around each fragment and the ends brought out through small skin incisions and attached to metal bars (coat hanger wire) coming down from a plaster of Paris head cap (Fig. 19). By this means the fragments were held in normal relationship. Fig. 20 is from a radiograph made at this time and shows the circumferential wires and also the gap created between the fragments after they were properly reduced by the osteotomy. After waiting for about six weeks for healing from this preliminary operation, the gap in the mandible was filled with a bone graft from the crest of the ilium

Fig. 13 (Case 4).—Extensive brown hairy nevus of left side of face.

Fig. 14 (Case 4).—Tube pedicle flap on abdomen with one end attached to wrist for later transfer to face.

Fig. 15 (Case 4).—Tube pedicle flap from abdomen carried to left side of face on right wrist.

Fig. 16 (Case 4).—Result after excision of growth and covering of raw area with tube pedicle flap from abdomen.

(Fig. 21). Consolidation took place in eight weeks, the circumferential wires and headcap were removed, and the patient was then able to have full upper and lower artificial teeth made, with which he could masticate (Fig. 22).

Case 6.—In this case is illustrated a unilateral defect of the mandible with retrusion of the chin. The patient in this case was an 18-year-old boy, who, when 8 years of age, had had a resection of several inches of the left side of the mandible for a tumor, reported to be a sarcoma. Later, two attempts were made in another city to restore the defect in the mandible by rib grafts, which were unsuccessful. Examination showed the typical deformity resulting from a defect of one side of the mandible. There was marked shortening on the left side, causing the large segment of the lower jaw, with its teeth, to be drawn over toward the left, attached by fibrous tissue to a short left segment consisting of the ascending ramus and the coronoid and condyloid processes. There were no teeth on the left side beyond the first incisor, and the remaining mandibular teeth had no relationship with the upper teeth, being drawn backward and inward. He could open his mouth widely, but there was a marked deviation of the lower jaw to the left (Fig. 23). The profile showed marked recession of the chin. There was a dense external scar adherent to the bone on the left side (Figs. 24 and 25). Fig. 26 is from a radiograph showing preoperative condition of the bone on left. Correction of this case involved several steps:

1. Division of fibrous adhesions between the ends of the mandibular fragments and remains of the rib graft on the left side to allow the main portion of the lower jaw and teeth to be carried to the right and forward into more normal position. After the adhesions had been severed it was possible to bring the lower teeth into fairly good occlusion with the upper and hold them there by means of arch wires. This interdental fixation was supplemented by a circumferential wire around the symphysis, forward traction on the chin being maintained by attachment of the wire passing around the bone to a bar coming down in front of the face from a plaster of Paris headcap. This forward traction was maintained for about three weeks, after which fixation was continued by the wires attached to the teeth.

2. Replacement of the dense scar over the operative field by good skin and subcutaneous tissue. This was accomplished by transplantation of a tube pedicle flap from the left clavicular area.

3. Restoration of the length and continuity of the left side of the mandible by a bone graft. Three months after the first operation, the wounds from these preliminary operations having healed, a graft two and one-half inches in length and comprising the entire width of the crest of the left ilium, was removed with a metacarpal saw, and after exposure of the ends of the separated mandibular fragments, it was inserted in the gap, being fastened to each fragment by brass wire sutures passed through holes drilled in the bone (Fig. 27). This operation was followed by some low-grade suppuration, which stopped after removal of one of the suture wires two months later. Shortly after this, the wire fixation on the teeth was discontinued, and union of the graft with stabilization of the jaw was found to be complete. Figs. 28 and 29 show the occlusion of the teeth at that time and also the mouth opening, with better central position of the mandible.



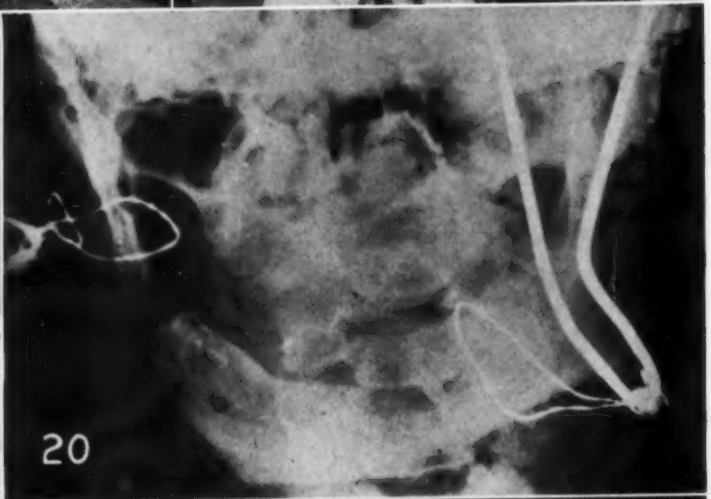
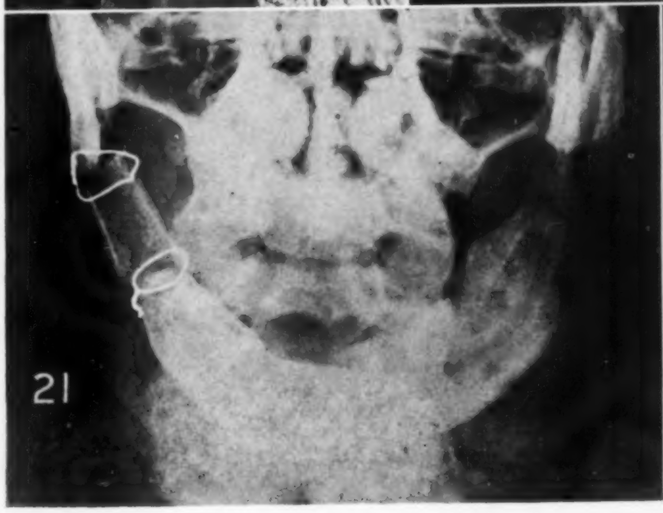





Fig. 17 (Case 5).—Deviation of chin to left due to malunion of fracture of left ramus of mandible.

Fig. 18 (Case 5).—Restriction of opening of mouth and malrelationship of edentulous upper and lower alveolar arches rendering impossible insertion of artificial dentures.

Fig. 19 (Case 5).—Fixation of fragments after osteotomy by circumferential wires and plaster headcap.

Fig. 20 (Case 5).—Radiograph after osteotomy showing gap in bone on left side and circumferential wires.

Fig. 21 (Case 5).—Radiograph showing gap in mandible filled with graft from crest of ilium, bringing about stabilization and restoration of length.

Fig. 22 (Case 5).—After restoration of correct position of jaw and increase in mouth opening, permitting insertion of artificial dentures.

4. Additional prominence to the chin and restoration of occlusion by provision of artificial lower anterior teeth. Four months after the bone-grafting operation, the mucous membrane in the vestibule of the mouth beneath the anterior mandibular teeth was divided by an incision extending from one premolar

Fig. 23 (Case 6).—Marked deviation of lower jaw to left on opening mouth due to loss of bone on left side.

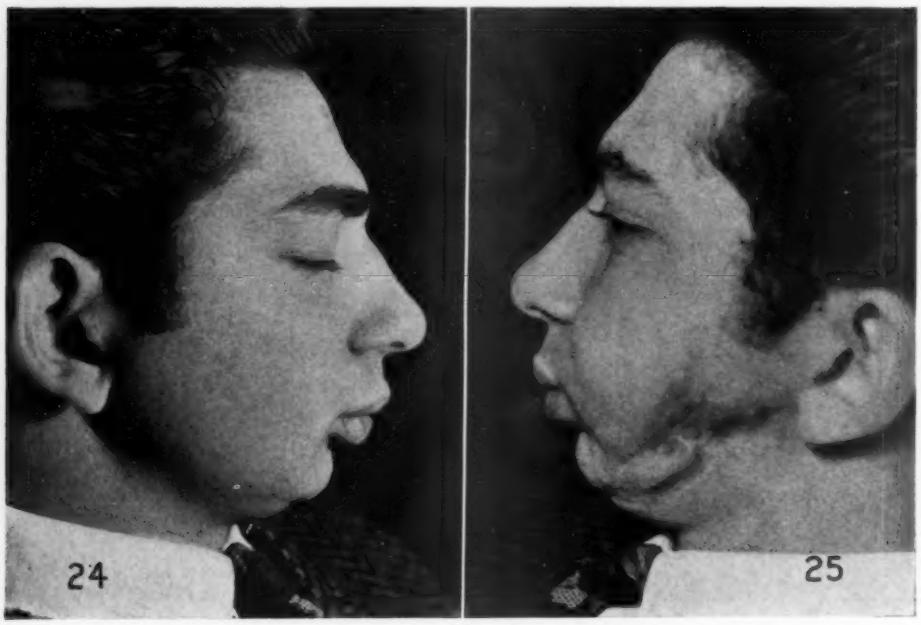


Fig. 24 (Case 6).—Right profile before treatment showing backward position of chin.
Fig. 25 (Case 6).—Left profile before treatment showing adherent scar.

region to the other, and the soft tissues were separated from the periosteum over the anterior surface of the mandible almost down to its lower border. This created a deep pocket in the soft tissues behind the lip, which was then lined with

a split-skin graft supported on a mold of dental impression compound. This was held in position for several days by circumferential sutures. After removal of the compound, the pocket was found to be lined with skin (Fig. 30). The patient

26

27

Fig. 26 (Case 6).—Radiograph showing defect in left side of mandible and remains of unsuccessful rib graft.

Fig. 27 (Case 6).—Radiograph showing restoration of continuity and length of left side of mandible by graph from crest of ilium.

then had a partial denture* constructed of acrylic material, which fitted over the natural lower anterior teeth and which occluded well with the upper teeth. An extension from the appliance passed down into the skin-lined pocket to build out

*Dr. Frank A. Fox, University of Pennsylvania Dental School, constructed the denture.

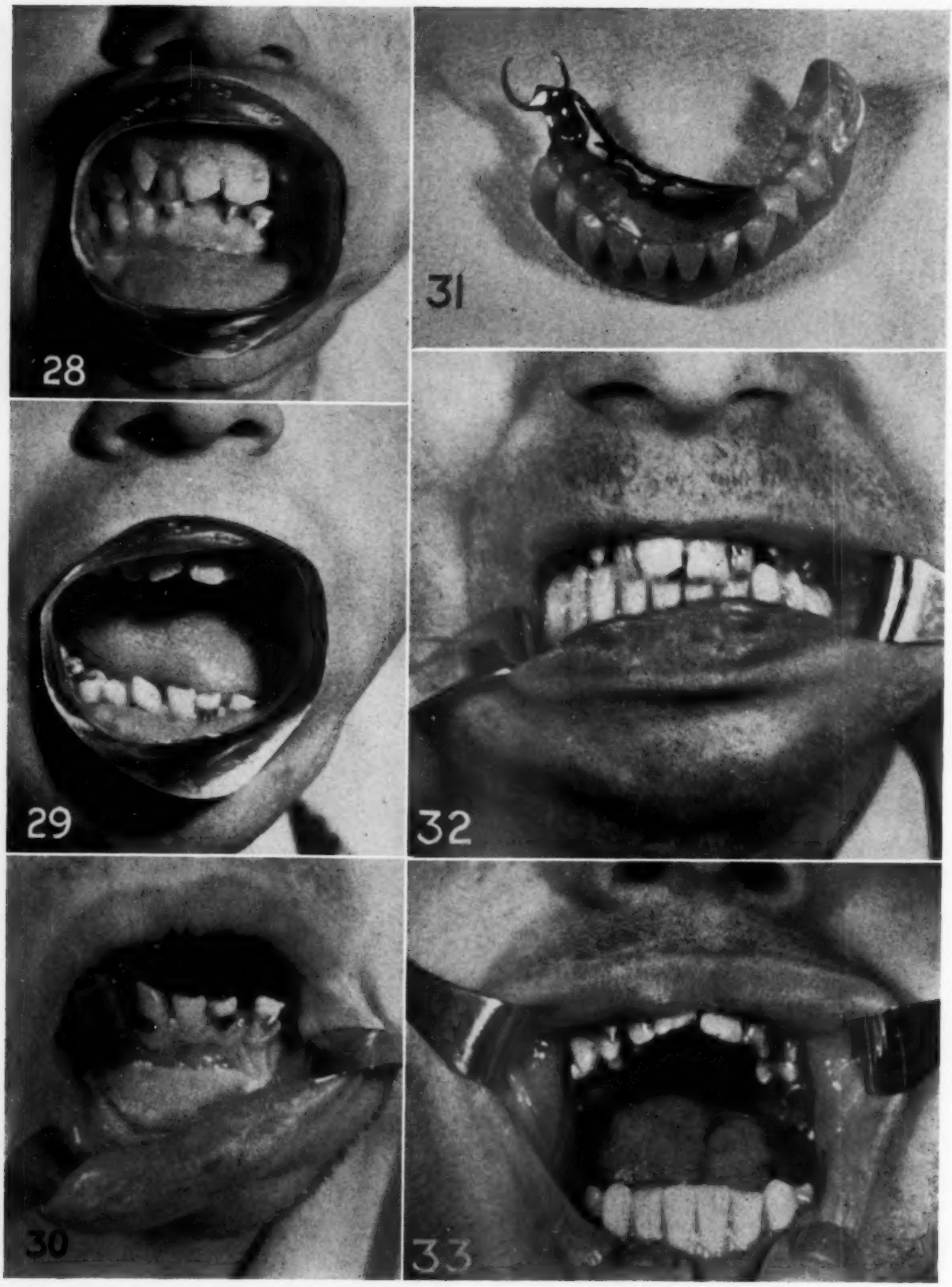


Fig. 28 (Case 6).—Improvement in occlusion of teeth after bone graft.

Fig. 29 (Case 6).—Disappearance of deviation of mandible to left on opening mouth after stabilization of jaw by bone graft. Compare with Fig. 23.

Fig. 30 (Case 6).—Pocket between bone and soft tissues of chin lined with split-skin graft.

Fig. 31 (Case 6).—Artificial appliance to supply missing teeth and build out prominence of chin (Courtesy of Dr. Frank A. Fox).

Fig. 32 (Case 6).—Artificial appliance in place, mouth closed, showing good occlusion of teeth.

Fig. 33 (Case 6).—Artificial appliance in place, mouth open.

Fig. 34 (Case 6). —Improved profile after completion of treatment. Compare with Fig. 24.

Fig. 35 (Case 7).— Extensive radiation necrosis of right malar and maxilla with destruction of soft tissues of cheek.

Fig. 36 (Case 7).— Opening through right cheek into maxillary sinus and nose after removal of dead bone. Tube pedicle flap from axilla to upper part of chest, to be used later for repair of openings in face and mouth.

the chin to a considerable extent. The patient's jaw is now stable, with normal opening and closing, and with the appliance in place he is able to masticate any kind of food; also, the profile is greatly improved (Figs. 31, 32, 33, and 34).

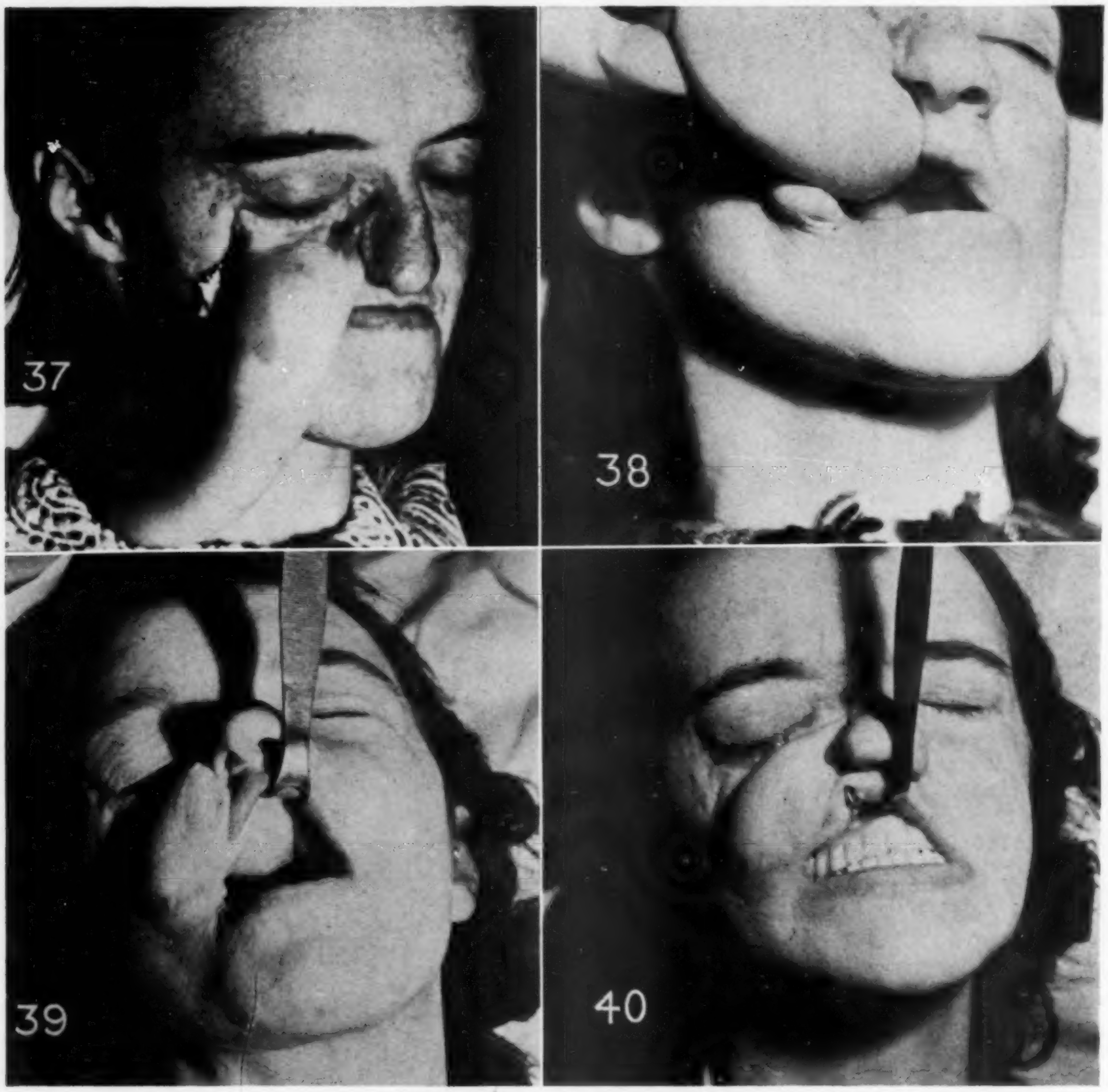


Fig. 37 (Case 7).—End of tube pedicle from chest sutured to cover opening in cheek.
Fig. 38 (Case 7).—Other end of tube pedicle sutured to edges of defect in hard palate.
Fig. 39 (Case 7).—Defect in hard palate completely closed by skin flap.
Fig. 40 (Case 7).—Artificial teeth inserted.

Case 7.—In this case is illustrated a defect of the cheek and palate resulting from radiation treatment for carcinoma of the upper jaw. A female, single, aged 25, had been first treated three years previously by x-rays and radium for a lesion of the upper right jaw, diagnosed as carcinoma of the right maxillary sinus, verified by biopsy. As a result of the treatment, the soft tissues of the cheek sloughed, exposing the necrotic malar and maxillary bones on the right side (Fig. 35). No evidence of the original malignancy remained, but the surrounding skin showed marked atrophic changes. At the first operation a large seques-

trum was removed, comprising most of the right malar and maxilla including the palatal and alveolar processes, and the teeth attached to it, as well as the septum of the nose. This left a large opening in the right cheek, into the maxillary sinus and nose, and a defect comprising the entire right side of the hard palate. This caused marked impairment of speech. At the time the dead bone was removed, a long tube pedicle was prepared from the right anterior axillary fold down to the groin to be used later to repair the face. Fig. 36 shows an intermediate transfer of the tube pedicle to the upper part of the chest and the defect in the cheek. After the first operation it became necessary to wait several months before attempting repair of the facial defect, until all infection had cleared up and remaining dead bone had come away. Pathologic examination of tissue removed from the edges of the defect and from the cavity from time to time showed no persistence of the malignancy. Eight months after the first operation, the distal end of the flap was severed from the axillary region and,

Fig. 41 (Case 7).—Present external appearance, subject to further improvement by thinning out of flap, smoothing of edges, etc.

after freshening the edges of the opening in the cheek, the end of the tube was flattened out by excision of intervening fat to create two surfaces of skin, one being sutured to the edges of the defect facing in to replace the lining of the maxillary sinus and the other facing out to replace the lost external skin (Fig. 37). Considerable difficulty was experienced in getting the skin of the flap to heal well to the poorly nourished skin surrounding the defect. Four months elapsed, therefore, before it was considered safe to proceed with the next stage. Then, the flap was cut loose from the chest and the free end was in turn thinned out to form two skin surfaces, which were then sutured into the palatal defect, forming upper and lower layers. In order to introduce the end of the flap into the mouth it was necessary to divide the right side of the upper lip, and this was later repaired. Fig. 38 shows the tube, forming a loop, with one end on the cheek

and the other in the palate. After a few weeks it was noted that the opening in the palate had been completely closed by the skin flap. The loop was then divided where it emerged from the mouth. The freshened ends were trimmed to fit the remaining free edges of the cheek and mouth defects, respectively, and sutured in place. Later, several minor operations were done to thin out and shape the cheek flap, and a piece of costal cartilage was inserted beneath the lower eyelid to support the right eyeball. An upper artificial denture* was made, enabling the patient to masticate food with satisfaction. The marked defect in speech was immediately overcome by operative repair of the palate, and her appearance was greatly improved (Figs. 39, 40, and 41).

*See footnote on page 89.

A COMPARISON IN METHODS OF PREVENTION OF LOCAL ALVEOLAR OSTEITIS

L. T. Russell, Jr., D.D.S., Charlotte, N. C.

IT HAS long been my opinion that, aside from lowered tissue resistance produced locally by trauma in the extraction of teeth, the ingress of mouth fluids into the fresh wound of a tooth socket is principally responsible for the condition known commonly as dry socket (local alveolar osteitis). I do not mean that the saliva per se is the etiological factor in producing alveolar osteitis, but the fact that the saliva, even in the most healthy and hygienic mouths, is infested with a variety of pathogenic organisms cannot be overlooked. These organisms, particularly the low-grade pyogenic variety, I believe to be the actual etiological factor in producing dry socket.

In expressing this opinion, I am not forgetting that trauma is a definite factor in certain cases, because it is a proven fact that trauma produces lowered local tissue resistance, and, if it is severe enough, actual death of the tissues will ensue. Also, I am mindful of the presence of the preoperative infection, which is, after all, the reason for extraction in the majority of cases. It must be remembered, however, that this infection, existing before extraction is performed, is usually of a chronic type and the defense mechanism of the body has become activated by the very onset of the infection in an effort to cope with the situation. Usually the inflammatory reaction produced is of a mild character, depending, of course, on the degree of severity of the initial infection.

Over a period of time, this mild chronic inflammatory process succeeds in building a limiting or protecting zone or "walling off" of the infection, which protects the bone from further invasion. This limiting zone (healthy granulation) also more or less protects the nerve ending in the area, which, to me, explains why, in so many cases of definite long-standing infections, we do not get painful dry sockets, despite the fact that the central portion of the infected area consists of unhealthy granulations (granuloma). With this condition present, the devitalized tooth in position acts as a constant irritant, and the process of inflammation is a continuous one. Such factors as lowered bodily resistance or excessive pressure on the tooth in biting, or both, may cause the chronic condition to become acute at any time.

Let me say here that extraction in acute conditions is definitely contraindicated, unless there is actual clinical indication that pus is present around the roots of the tooth and that drainage might be established by extracting the tooth. In the majority of such cases, I find that bleeding is slight, and, with open and free drainage, healing is rapid and without complications.

In cases where bleeding is as we normally expect, the formation and maintenance of a healthy blood clot are essential to proper healing. The maintenance of a healthy blood clot is my aim in using silver foil. By covering the socket with silver foil immediately following extraction, we can, to a great degree,

seal the socket against the entrance of bacteria-infected saliva, which, I believe, will prevent subsequent disintegration of the blood clot and infection of the alveolar socket.

In the following clinical experiment and observations, I have omitted extractions involving radical surgery, or cases which would naturally necessitate more than the ordinary amount of trauma, such as impactions or extractions which are classed as difficult.

In one hundred cases of simple extraction I have employed silver foil as a means to prevent the saliva, as much as possible, from entering fresh socket wounds. Also, in one hundred cases of simple extractions I have used one of, or a combination of sterile gauze sponges over the socket and pressure, for ten minutes. In one hundred other cases of simple extraction I have used only the sterile gauze sponges (as control).

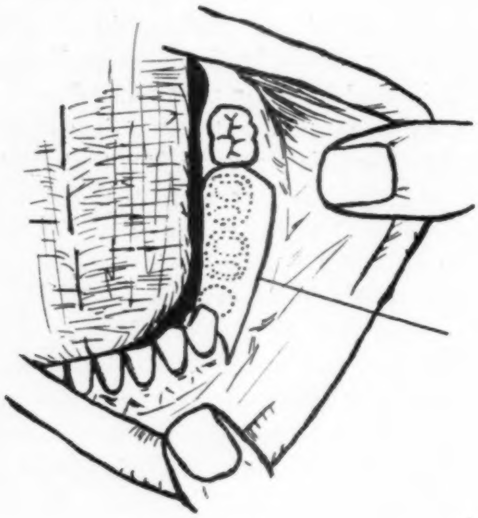


Fig. 1.

In carrying out the experiment, I made no choice in patients or extractions; starting with the first as outlined above, every third case became a control.

Observations are as follows:

1. Control (gauze sponge only)	14 cases of osteitis out of 100 (5 severe)
2. Sulfa drugs	9 cases of osteitis out of 100
3. Silver foil	3 cases of osteitis out of 100 (all mild)

Technique.—Before extraction, cut a small strip of silver foil (adhesive) to the approximate width of the space to be left when the tooth is removed. This width should be the mesiodistal measurement of the crown of the tooth (approximately). The length of the strip should be determined by the distance from the buccal or labial sulcus molded across the ridge and ending just short of the floor of the mouth.

Immediately following extraction, mop blood and saliva from the area with sterile sponge; in so doing you will dry the mucosa sufficiently to permit the foil to adhere properly.

The foil, having been properly cut to size, is placed around the bulb of the index finger (adhesive surface outward) and carried to the socket. With the other hand use either finger or instrument to mold the foil to place (Fig. 1). Then, with the index fingers of both hands, hold the foil in place for a few seconds. Rinse the mouth and dismiss the patient, after cautioning him to refrain from eating or otherwise displacing the foil for two hours. After six hours, a warm saline mouthwash may be used at two-hour intervals.

418 PROFESSIONAL BUILDING

MOUTH LESIONS OF INTEREST

Neal W. Chilton, B.S., D.D.S.,* New York, N. Y.

TUBERCULOSIS OF THE TONGUE

A PUERTO RICAN male of about 32 years of age was in the last stages of Hodgkin's disease in the medical ward of the hospital. The disease was of long standing, and attempts to treat it by x-ray emanations had produced an aplastic anemia. We were called to look at the patient who, although apathetic and dying, was complaining of bleeding from the mouth.

Examination.—When the patient was examined, no oral bleeding was noted, but bilateral painful ulcers were observed on the tip of the tongue. The lesions had a granular grayish-yellow appearance with a reddish punched-out center, resting on a circumscribed, slightly raised, smooth red area about the size of a 10-cent piece. Except for a coating of the dorsum of the tongue and a dry scaliness of the lips, usually present in long-standing debilitating disease, the mouth was otherwise negative.

Fig. 1.—Tuberculosis of the tongue.

Course.—In spite of the fact that there was no history of tuberculosis, the author described the lesions on the hospital chart as being tuberculous in nature. Since the patient was obviously beyond hope and under adequate sedation, no treatment (beyond the usual 1 per cent gentian violet swab) was instituted. He died three days after the lesions were noted. Post-mortem examination revealed

*From the Dental Division, Lincoln Hospital, M. H. Feldman, Chief.

the presence of miliary tuberculosis as well as Hodgkin's disease. Unfortunately, microscopic examination of the tongue lesions was not performed. (The author was not informed of the autopsy until after it had been performed.) A diagnosis of tuberculosis of the tongue was made.

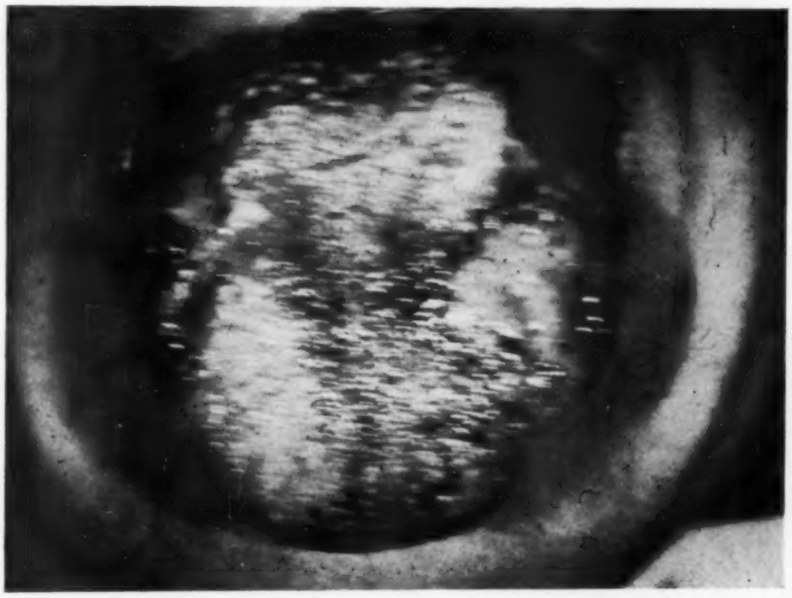


Fig. 2.—Phenobarbital stomatitis (tongue).

Fig. 3.—Phenobarbital stomatitis (cheek).

PHENOBARBITAL STOMATITIS

A 32-year-old white female patient came to the Dental Clinic complaining of severe burning pain in the mouth and tongue of two days' duration, which

prevented her from eating or swallowing. Upon interrogation, it was found that four days previously she had started taking elixir phenobarbital for "nervousness," upon a physician's advice. Two days after she began the use of this drug, the oral condition manifested itself.

Examination.—The lips, tongue, and buccal mucous membrane were the only parts of the oral cavity affected. The inner surface of both cheeks appeared a very angry red with evidence of desquamation. Irregular flattened areas of whitish-yellow patches were present on these surfaces. Attempts to wipe the area produced intense pain and bleeding. The vermilion border of the lips was slightly scaly. The tongue was the angriest the author had ever seen. Whitish areas of papillar hypertrophy appeared on the dorsum, while completely denuded red areas appeared on the sides and tip as well as the dorsum of the tongue. There were no lesions on the body.

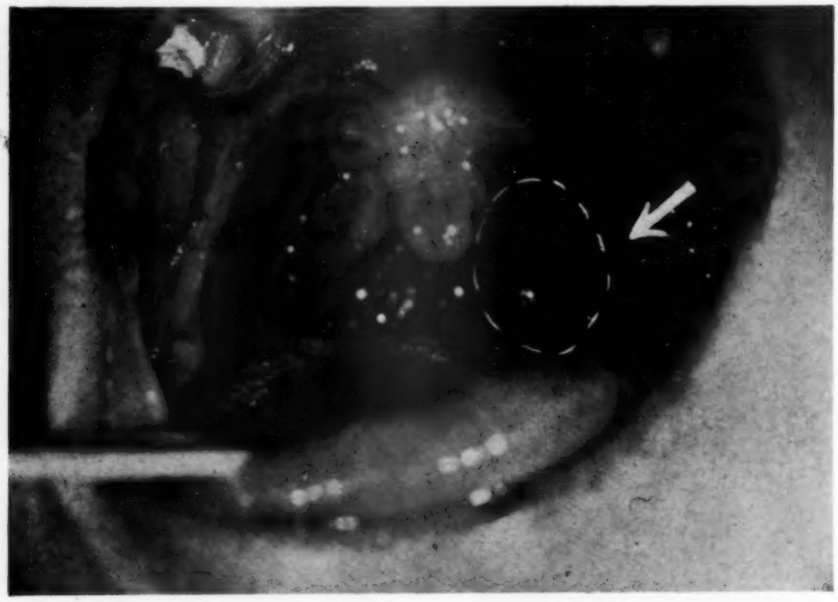


Fig. 4.—Vincent's angina (dotted area).

Treatment.—The patient was advised to coat her tongue with vaseline, drink dilute fruit juices, and partake only of a bland diet. A solution of $KMnO_4$ (1:5000) was prescribed as a mouthwash three times a day. A tentative diagnosis of phenobarbital stomatitis was made and the patient was instructed to discontinue the use of the barbiturate. At the Dermatology Clinic, where I brought the patient for consultation, the diagnoses varied from beginning pemphigus, to idiopathic stomatitis, to phenobarbital stomatitis.

Course.—The patient was next seen four days later; the lesions and symptoms had completely disappeared. Further attempts to determine whether the patient was allergic to phenobarbital were unsuccessful due to lack of cooperation on the part of the patient. A final diagnosis of phenobarbital stomatitis (dermatitis venenata) was made.

VINCENT'S ANGINA OR FOLLICULAR TONSILLITIS

A member of the hospital staff, a 24-year-old white woman, was complaining of sore throat, difficulty in swallowing and a slight malaise. This progressed until the patient was unable to swallow, had a great deal of pain in her throat, and was confined to bed.

Examination.—I saw the patient on the fifth day after the onset of the condition. Examination of the throat revealed an injected pharynx and a livid red left tonsillar area. This area extended to the left and behind the uvula to the anterior faucial pillar. Tonsils were present on both sides. A very deep crypt divided the left tonsil into two lobes. Attached to this "crevice" was an area, about the size of a five-cent piece, of a dark greenish-yellow appearance, which was difficult to remove. The perpihery of this ulceromembranous area was yellow, on a red base, and its center appeared grayish green. The left submaxillary lymph nodes were markedly enlarged. At this time the patient's temperature was 102.8° F. The gingivae and oral mucosa appeared normal. Except for a slight malaise and the fever already noted, the patient exhibited no systemic manifestations.

Treatment.—The left tonsillar area was swabbed with 3 per cent hydrogen peroxide twice during the third day and then discontinued. She received intramuscular injections of vitamin B_1 (2 c.c. every other day) and oral doses of vitamin C (150 mg. per day for the last five days). The throat was irrigated with hot saline solution three times a day.

Course.—The condition lasted for seven days and then completely disappeared. The patient experienced no further discomfort and went about her hospital duties as before.

From the clinical appearance alone (no smears were taken), the chief of the Ear-Nose-Throat Division made a diagnosis of Vincent's angina.

Opinion, however, was divided on the diagnosis. The medical resident believed that it was a follicular tonsillitis. He based his opinion both on the appearance of the lesion and the fact that swabbing almost continuously with Durante's solution for the first two days did not have any beneficial effect. The condition became worse, causing the patient to be confined to bed. The author tends to concur with this diagnosis.

Case Reports

CASE NO. 88

HYPOPLASIA OF THE DENTITION

Harry C. Brown, D.D.S., Bloomington, Ill.

X-RAYS of the dentition of a male patient 19 years of age are presented. His father's teeth were about the same; he lost them at an early age and is wearing dentures. The dentist who extracted the teeth made the statement that most of them could be removed with little effort, and some were exfoliated during mastication. According to the family physician, both father and son enjoy good physical health.

The father's brother was completely toothless. This appears, therefore, to be a modification of anodontia, a congenital, hereditary sex-linked, recessive disease, generally associated with other signs of ectodermal dysplasia.

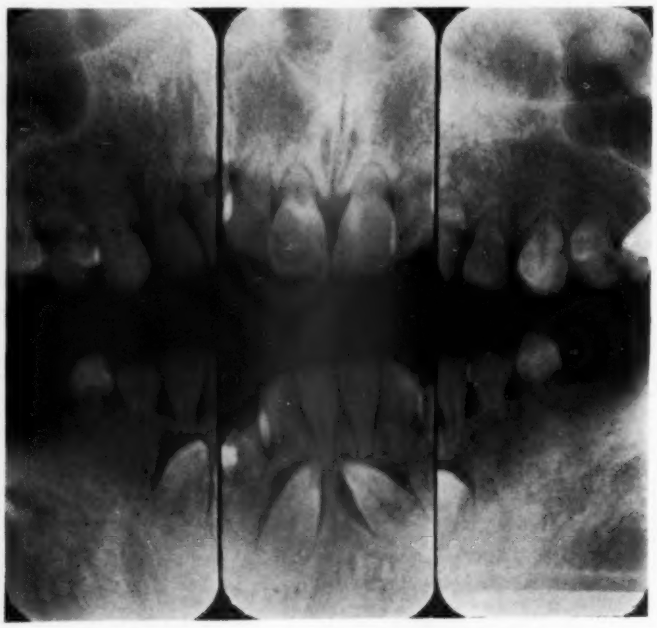


Fig. 1.

X-ray examination shows that the patient has developed no third molars in the upper jaw, and the right second premolar is missing. In the mandible there are only three permanent molars present, the canines are unerupted, and

it is difficult to state whether the premolars are permanent teeth or whether they are deciduous molars.

The striking feature of the boy's dentition is the marked underdevelopment of the individual teeth, particularly the roots, which are stunted in growth. This accounts for the spontaneous shedding and easy removal of the father's teeth. An interesting feature is the comparatively large size of the mandibular canines. This is in accord with the findings that canines are, in both the human and mammalian dentition, most persistent in their development. Another observation that may be pointed out is the dense sclerotic bone around the short roots, particularly in the maxilla.

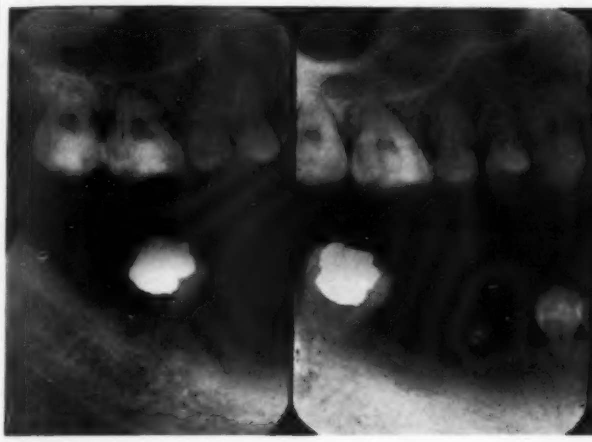


Fig. 2.

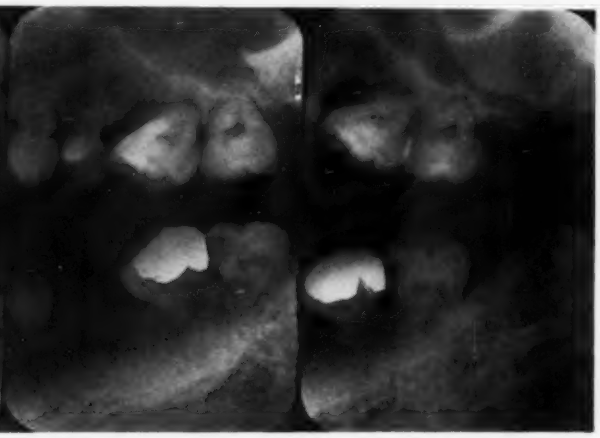


Fig. 3.

A patient has been presented with marked weakness in the tooth-forming epithelial structures, combined with a deficiency in the mesodermal components of the tooth germ, resulting in stunted roots. The condition has a hereditary history, the patient's father having an identical dental deficiency, and his uncle having complete anodontia.

UNITY BUILDING

CASE NO. 89

DISPLACED THIRD MOLAR WITH FOLLICULAR CYST

Harry J. Field, D.D.S., and Alfred A. Ackerman, A.B., B.Sc., D.D.S.,
Newark, N. J.

Chief Complaint.—Intense pain and swelling lower right side accompanied by severe trismus and rigidity.

Radiographic Examination.—A small intraoral film revealed a normal second molar with third molar apparently absent. A suspicious radiolucent area may be seen at the anterior border of the ramus on this film (Fig. 1).

Fig. 1.

Fig. 2.

A lateral plate of the jaw revealed the presence of the displaced third molar high in the ramus. A large follicular cyst surrounded the tooth (Fig. 2).

Surgery.—As a first measure, to provide relief from pain, intraosseous drainage was necessary. A large opening, 1 cm. in diameter, was made through the anterior wall of the ramus into the cyst cavity. A large quantity of pus was evacuated. Due to the severe rigidity of the jaws, the removal of the unerupted third molar and the enucleation of the cyst were postponed for ninety days. At that time, the trismus had completely disappeared and the surgery was completed. The membranous wall of the cyst was thick and easily enucleated from the bone walls. The entire anatomic contents of the mandibular canal were clearly discernible following removal of cyst membrane. None of these structures was injured, as evidenced by the fact that there was no paresthesia following the surgery.

Comments.—We wish, first, to demonstrate the importance of lateral views of the jaw whenever suspicious shadows are seen on the small dental films. Also, this case again clearly illustrates the importance of early drainage in acute infections of cystic lesions even if such drainage can be effected only by going through the bone to reach the pus.

130 MARKET STREET

Editorial

Penicillin

Major Champ Lyons writes in the *Journal of the American Medical Association** that on April 1, 1943, the office of the Surgeon General, U. S. Army, sponsored a pilot unit for penicillin therapy at the Bushnell General Hospital, Brigham City, Utah, and in June, at the Halloran General Hospital, Staten Island, New York. These, he stated, have served as "schools" to train selected medical officers in the use of the drug in surgical infections. Lyons then reviewed in his paper: the methods of administration, dosage, and reactions; the experience in the treatment of acute pyogenic infections; and the experience in the treatment of chronically septic compound fractures.

Penicillin may be administered by either the intravenous or the intramuscular route, the latter having proved more practical than the former. No contraindication to its continued use has been observed. For bacteremic infections, the constant intravenous treatment is recommended. The susceptibility of bacteria to penicillin is variable, not only from group to group but from strain to strain, and, therefore, it is recommended to maintain laboratory controls of the potency of penicillin and bacterial susceptibility, to insure uniformly successful results. The dose recommended is about 90,000 units daily for streptococci and similarly sensitive bacteria; 200,000 to 400,000 units daily for staphylococcic infections, given intramuscularly, in divided doses every three hours in concentrations of 5,000 units per 1 c.c. Local therapy may be used in a supplemental manner in wounds appropriate for such treatment; more dilute solutions containing 250 units per cubic centimeter have been found satisfactory by the investigator.

Penicillin, however, is not effective in all types of infection. The writer concludes that penicillin is effective as an efficient antibacterial agent "in the treatment of acute infections caused by staphylococci, hemolytic and nonhemolytic streptococci, mixed infections due to gram-positive bacteria, and actinomycosis. Gram-negative diplococci are susceptible, but gram-negative bacilli are resistant, to treatment. Mixed infections with both gram-positive and gram-negative bacteria may be benefited through the effect on the susceptible bacterial species."

For septic fractures, defined as putrid wound infections, Lyons states "staphylococci and streptococci are rapidly responsive to therapy. Anaerobic cellulitis, due to proteolytic bacteria of putrid wound infections, responds to penicillin, but the bacteria may persist in the presence of devitalized tissue or wound exudates."

It should be pointed out that penicillin treatment is to be looked at as a supplemental therapy in an over-all surgical program. Surgical intervention should be carried out, and, in cases of osteomyelitis, sequestra and foreign bodies should be removed, as they are frequently the cause of recurrence of infection following

*J. A. M. A. 123: 1007, 1943.

prompt improvement; "penicillin is unable to sterilize such foci of infection." Supportive treatment should not be omitted. Lyons stresses the importance of correcting reduced blood volume, a deficiency of the total circulating and available hemoglobin, and an excessive interstitial fluid volume. The best supplemental source of hemoglobin is whole blood, which should be given by transfusion.

The editor has had only one experience with penicillin therapy. It was a case of a septic compound fracture at the angle of the jaw, which resisted all forms of local therapy by the best conventional methods, including dakinization. The mixed infection responded after five days of penicillin therapy, 10,000 units being administered intramuscularly every three hours. The α hemolytic streptococcus disappeared first, after five days of treatment, the hemolytic *Staphylococcus aureus* after seven days. The gram-negative bacillus Bacteroides, and *Clostridium sporogenes* were the contaminants; the latter disappeared almost immediately, but the former proved quite resistant. On the eleventh day, both the streptococcus and staphylococcus had reappeared, together with a new organism, the *Clostridium welchii,* which was removed on the seventeenth day of treatment, the streptococcus having been lost on the fourteenth day. The clinical picture was dramatically improved; the profuse pus discharge was almost completely arrested. However, the staphylococcus persisted, although it remained sensitive to penicillin. The cause was believed to be a small sequestrum, which was located by x-ray examination.

K. H. T.

Abstracts and Reviews

Oral Diagnosis, With Suggestions for Treatment: By Kurt H. Thoma, D.M.D., Professor of Oral Surgery and Professor of Oral Pathology, Harvard University, Oral Surgeon and Chief of Dental Department, Massachusetts General Hospital, ed. 2, Philadelphia and London, W. B. Saunders Co., 1943.

This book is a revision of the original *Oral Diagnosis and Treatment Planning.* The present book now represents a text planned for use as a teaching aid for students and teachers in courses in oral diagnosis. There has been a complete reorganization of the index, which now lends itself to orderly reference. The index has been detailed so that any abnormality of the oral cavity alone or in association with other structures or diseases may be easily found.

There is excellent detailed description of acceptable methods for clinical and laboratory examination. The interpretations are based upon the author's wide experience as well as on that of his associates. It is surprising to see so much information concerning a portion of the body catalogued in a single text.

The etiological factors described for disturbances of the oral cavity may be found lacking in some quantity. This is no fault of the book, but merely represents our state of knowledge at the present time.

The text should be extremely valuable to dental students and medical students as well as to practitioners of dentistry and medicine.

D. W.

Treatment of Diseases of the Gums: By Capt. J. J. D. King. Brit. D. J. **75**: 197, October, 1943.

This article represents part of a symposium on the "Diagnosis and Treatment of Diseases of the Gums" at the annual meeting of the British Dental Association. The author restates the case for multiple causes for gingivitis and reviews some of the accepted nutritional factors. In the discussion following the article many practical clinical observations are viewed, concerning disease of the gums, and, especially, a logical approach to the study and treatment of Vincent's infection.

D. W.

MARCH, 1944

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Original Articles

BONE GROWTH AND PHYSIOLOGIC TOOTH MOVEMENT

HARRY SICHER, M.D., AND JOSEPH P. WEINMANN, M.D.

INTRODUCTION

PHYSIOLOGIC tooth movement and bone changes in the jaws are inseparably associated phenomena. To decide which of the two is primary is of greatest interest not only from a theoretical but also from a practical viewpoint. Investigations on human material and on isolated groups of teeth (molars of rats) have not been able to show heretofore more than the actual association of bone changes with movement of teeth. A deeper insight can be gained only by correlating the findings on the growth of jaws and the simultaneous movements of teeth. The rat affords a very promising object for these studies because of the accumulation of a series of special anatomic features. These are:

1. The presence of continuously erupting and growing incisors.
2. The growth of the incisors in volume and weight throughout the life of the animal.
3. The presence of molars with limited growth which erupt in the first weeks of the animal's life.
4. The stability of the relative position of the incisors and molars to each other and to the growing jaw throughout the life of the animal.
5. The participation of two bones, the premaxilla and the maxilla, united by a suture, in forming the alveolus of the upper incisor.

These facts obviously complicate the changes in teeth and bone; an analysis of their interrelations, however, is for the same reason extremely revealing.

REVIEW OF LITERATURE

Human teeth drift, throughout the life of the individual, toward the midline. Apposition of bone on the distal alveolar wall and resorption on the mesial wall are characteristic for this physiologic phenomenon.[16]

Statements on the drifting movement of the rat molars are only casual and contradicting. Some authors[9, 11] speak of a mesial drift of the rat molars; one[10] reports a distal movement of these teeth.

The mesial drift of human teeth has been viewed as an adaptation to the loss of tooth substance at the contact areas from interproximal wear and as a tendency of teeth to keep in tight contact.[1, 16] Most authors see the moving force as the effect of masticatory pressure. It has been pointed out, for instance, that the human upper and lower molars are inclined mesially and, therefore, during mastication, exert pressure toward the midline.[3] The asser-

Foundation for Dental Research, Chicago College of Dental Surgery, Loyola University, Chicago.

tion that tooth eruption is subservient to alveolar bone growth[2] has been refuted by others.[9] Recent investigations have again strengthened the primacy of bone growth over tooth movement.[14, 15]

MATERIAL AND METHODS

The present study is based on the gross and radiographic analysis of skulls of albino rats from the Wistar strain ranging in age from 4 weeks to 1 year.* In addition, ground sections and decalcified serial sections of rat jaws cut in different planes were studied. Measurements were made on the skulls and on ground sections of animals which had received repeated injections of alizarin. Superposition of roentgenograms proved very useful in the study of age changes.

ANATOMY OF THE RAT DENTITION

In order to facilitate the evaluation of histologic findings and of certain mechanical theories of physiologic tooth movement, a detailed description of the rat dentition and of its relation to the bony structures is both necessary and desirable. This is the more important as the descriptions in the literature are incomplete. The paper of Addison and Appleton (*Journal of Morphology,* 1915) is restricted to a description of the incisor teeth only.

The rat has only four teeth in each quadrant: three molars and a single, very large incisor. The three molars of the rat have been frequently compared with human molars. The similarity, however, is more one in type than in detail. The second molars in the upper and lower jaw can best be taken as the starting point of description because they represent a rather simple pattern of a mammalian molar.

In the upper jaw the crown of the second molar is rhomboid with the longer diameter directed from mesiolingual to distobuccal (Fig. 1). Of the four cusps arranged on the corners of the rhombus, the distobuccal is the largest. The mesiobuccal and distolingual cusps are connected by a diagonal crest. A fifth accessory cusp is situated on the buccal edge between mesiobuccal and distobuccal cusps and buccal to both. The upper first molar can be visualized as formed by the addition of a strong mesial triangular lobe and cusp to the crown of the second molar. The third molar is a reduced image of the second. The mesiodistal diameter of the three molars decreases from the first to the third.

The upper second molar of the rat has four roots of almost equal thickness and length; the mesiolingual one is the strongest. The roots are arranged at the corners of a square (Fig. 6). The first molar has, corresponding to the additional mesial lobe of its crown, a fifth root which is by far the strongest. It is implanted obliquely to the crown and diverges quite considerably from the other four roots, of which the mesiobuccal is greatly reduced in length and diameter. The third molar has only three roots: two situated mesially, and the third, strong and flattened in a mesiolingual to distobuccal direction, situated distally.

The three upper molars are in close contact with each other and are separated from the incisor by a wide diastema. Their axes are inclined, causing

*We want to express our thanks to Dr. F. Herzberg and Dr. T. Schour of the University of Illinois Dental School, who kindly put the skeletal material of their collection at our disposal.

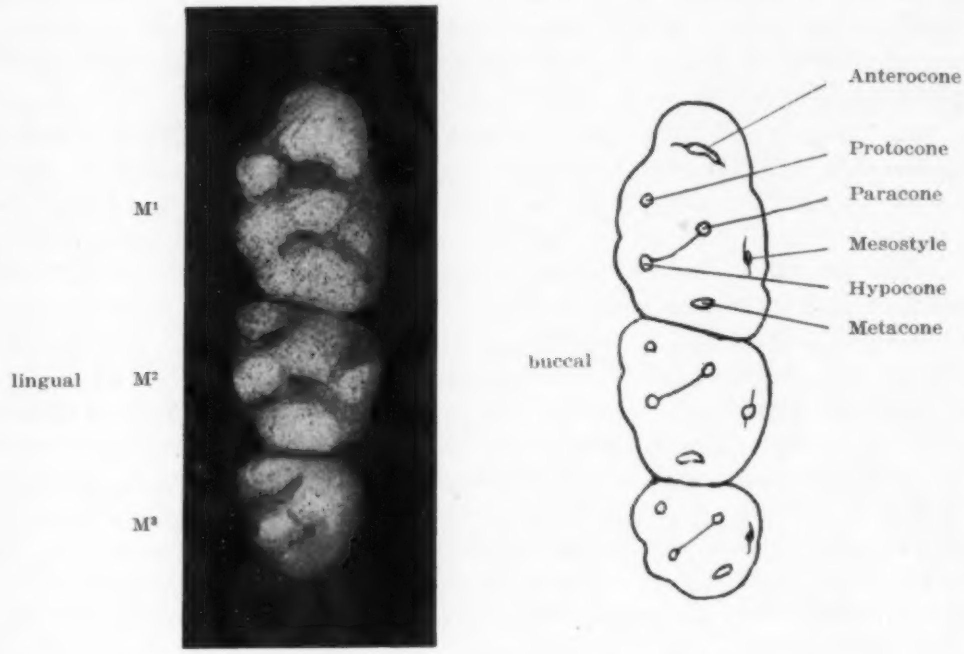


Fig. 1.—Left maxillary molars of a rat.

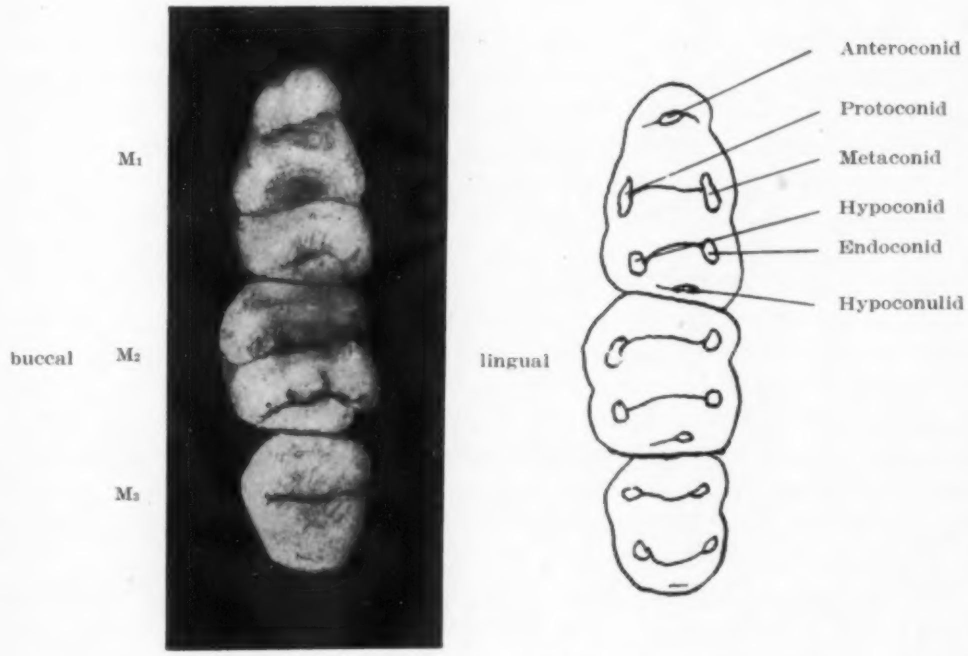


Fig. 2.—Left mandibular molars of a rat.

the crowns to point distally. The angle of this inclination decreases from the first to the third molar (Fig. 14). They are also inclined with the crown toward the buccal. Measuring across the entire upper arch, the distance between the right and left first molars is slightly smaller than that between the right and left third molars.

The lower second molar has five cusps, the fifth situated on the distal edge of the crown (Fig. 2). The two mesial and the two distal cusps are connected by strong transverse ridges. As in the upper jaw, the lower first molar can be derived from the second molar by the addition of a strong mesial lobe. The shape of the crown of the third molar is similar to that of the second molar. It is, however, reduced in size.

The lower second molar may have two or three roots (Fig. 7). If three roots are present, the mesiolingual is the smallest, the mesiobuccal of medium size, and the distolingual the largest. In most albino rats the two lingual roots fuse. Cross sections through the two roots in such cases are approximately bean-shaped; they are compressed from the mesiobuccal to distolingual. The first molar is four-rooted with two large roots situated under the mesial and distal extremities of the tooth and two thin, short roots occupying the space between mesial and distal roots, buccally and lingually, respectively (Fig. 7). The third molar has two roots in the same position as in a two-rooted second molar.

The inclination of the long axes of the lower molars is directed so as to point the crowns mesially (Fig. 12). The angle of inclination increases from the first to the third molar. In addition, the crowns are slightly inclined lingually. The distance between right and left molars is greater in the lower than in the upper jaw.

In occlusion the buccal cusps of the lower molars bite outside the buccal cusps of the upper molars. The fossa distal to the mesial lobe of the lower first molar articulates with the mesial lobe of the upper first molar.

The upper incisor is situated entirely in front of the molars (Fig. 14). Its curvature corresponds to a segment of a laterally sheared logarithmic spiral.[8] Its extra-alveolar part is curved backward, the attrition surface being almost vertical. The basal end of the incisor is found in the anterior part of the maxillary bone immediately above the bony plate of the hard palate (Fig. 16), lateral to the slit-shaped anterior palatine foramen (foramen incisivum). The thin alveolar bone of the basal end of the incisor bulges into the cavity of the maxillary sinus. At the basal end, the epithelial organ is bent to form the epithelial diaphragm.[14] The plane of the diaphragm slopes from above and in front, downward and backward. The fundic alveolar bone is parallel to the diaphragm, a fact which can easily be established by the comparison of sections and roentgenograms (Figs. 14 and 16).

The lower incisor is curved around a radius much longer than that of the upper incisor.[5] Its extra-alveolar part is directed anteriorly and upward; the attrition surface is inclined. The intra-alveolar part is found below the first molar, then laterally and below the second and third molar. The basal end is found in the ramus of the mandible above the level of the occlusal plane of the molars. The epithelial diaphragm and the fundic bone of the lower in-

cisor are inclined from down and in front, upward and backward (Fig. 12). This plane is parallel to a blunt crest on the medial surface of the ramus; this crest extends from the condyle anteriorly and inferiorly to the posterior end of the shelflike alveolar process of the lower molars (Fig. 5). This crista colli mandibulae (Sicher[13]) is a strengthening of the bone transmitting masticatory stresses to the condyle and to the base of the skull. At the same time the crest of the mandibular neck indicates the direction of the condylar growth.

GROWTH OF THE JAWS AND TEETH

Growth of the Upper Jaw.—The growth of the jaws in the rat follows the same general pattern as in man and other mammals. The upper jaw of the rat consists of two bones on either side, premaxilla and maxilla. They are joined to each other by the sutura incisiva or premaxillo-maxillary suture (Fig. 3). This suture starts on the lateral surface of the skull at the junction with the frontal bone, runs anteriorly and downward approximately in a sagittal plane, and then bends sharply back and down to reach the lower border of the upper jaw halfway between the incisor and first molar. From here it can be followed over the palatine surface of the upper jaw to the anterior palatine foramen (foramen incisivum) and then on to the nasal surface. Here it runs forward and upward to the frontal process and curves back to the frontal bone. The sites of growth of the upper jaw in length, height, and width are, besides the premaxillo-maxillary suture, the junction with the following bones: frontal bone, sphenoid bone, palatine bone, zygomatic bone, and the maxilla and premaxilla of the opposite side. In addition, there is apposition on the free alveolar borders and modeling apposition and resorption on the free surface of the bones. By its growth, the upper jaw shifts downward and anteriorly.

The growth in length occurs at the posterior end of the maxilla in the pterygomaxillary suture and between molars and incisor in the premaxillo-maxillary suture. The greater length of the maxilla necessitates a greater absolute growth increment than found in the mandible. The fact that the upper incisor lies entirely in front of the molars is the reason why the site of the additional increment in the maxilla is the premaxillo-maxillary suture. Thus, space is opened in front of the molars for the growing incisor.

Growth of the Lower Jaw.—The most important growth center of the mandible is the cartilage in the condyle,[4] which functions similarly to the epiphyseal cartilage of long bones. The growth is directed posteriorly and upward. At the same time the coronoid and angular processes enlarge by appositional growth at their posterior and superior margins (Fig. 4). Due to the condylar growth, the mandible moves anteriorly and inferiorly, the glenoid fossa of the temporal bone serving as fixed point. The growth of the mandibular body in height is almost entirely confined to apposition of bone at the alveolar crests and at the upper surface of the diastema between incisor and molars.

Growth of the Teeth.—After the formation of the roots, the growth of the molars is confined to lengthening and a slight thickening of the roots by apposition of cementum. The lengthening of the root partly compensates for the loss of tooth substance by attrition.[9] The growth of the incisors keeps pace

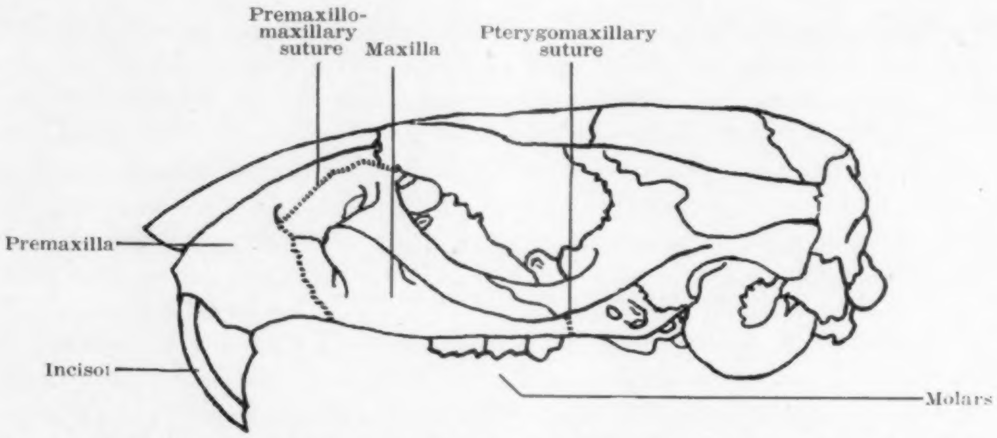


Fig. 3.—Lateral aspect of the rat skull indicating the sutures. Premaxillo-maxillary and pterygomaxillary sutures—dotted line. (Modified after E. C. Greene.[7])

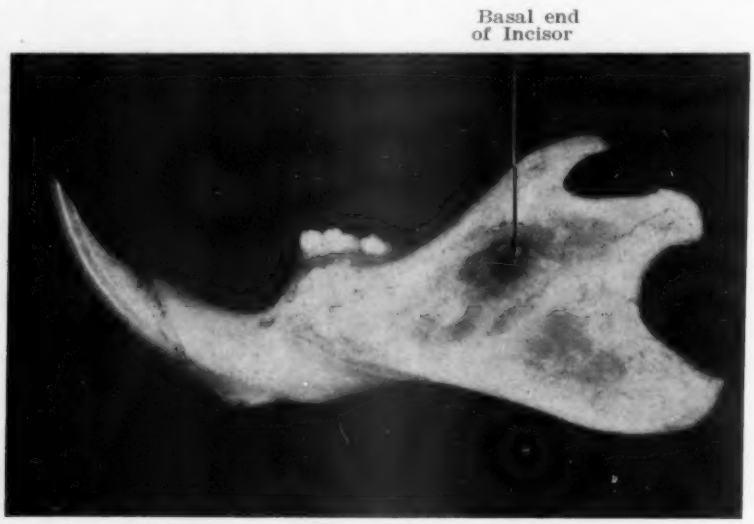


Fig. 4.—Lateral view of left rat mandible.

Fig. 5.—Medial view of left rat mandible.

with the general growth of the animal and the jaws. Here the growth is not only compensation for attrition, but also an absolute increase in size, as an adaptation to the enlargement of the masticatory apparatus. The incisor, as a continuously growing tooth, grows not only in length, but also in all its diameters and, therefore, in volume. Its curvature changes throughout the life of the animal, the radius of the curvature always increasing.

TABLE I

GROWTH CHANGES OF SKULL AND INCISORS. MEASUREMENTS IN MM.

AGE IN DAYS	LENGTH OF SKULL	DISTANCE FROM LAST LOWER MOLAR TO CONDYLE	DIASTEMA		EXTRA-ALVEOLAR LENGTH OF INCISOR		LABIOLINGUAL DIAMETER OF INCISOR	
			UPPER	LOWER	UPPER	LOWER	UPPER	LOWER
28	33.3	7.1	8.5	4.6	3.2	4.5	1.8	1.4
32	36.3	7.6	9.6	5.7	3.9	4.7	1.8	1.4
35	38.9	8.7	10.2	6.1	4.5	5.6	2.0	1.5
84	44.2	11.0	12.6	6.9	5.7	7.5	2.5	2.2
114	45.4	11.5	13.2	7.3	5.7	7.5	2.7	2.3
153	48.8	13.2	13.9	7.6	6.1	7.5	2.6	2.1
187	49.1	13.3	13.8	7.8	6.2	8.0	2.8	2.3
212	49.7	13.6	14.2	8.3	6.2	8.6	2.8	2.4
300	50.0	14.2	14.6	8.6	7.2	10.1	3.4	2.4

Measurements of the jaws show that, throughout the growth period of the rat, the position of the molars and incisors in the jaws remains practically constant (Table I).

MOVEMENT OF THE MOLARS

The molars erupt continuously.[9] At the same time, they move distally and slightly buccally. The direction and rate of movement can be ascertained by the observation of bone apposition and resorption. Ignoring for the moment the question of whether bone changes or tooth movement is the primary factor, the rate of bone apposition and the amount of newly formed bone is definitely equal to the rate and the amount of tooth movement. Horizontal sections cutting through all the roots of the three molars give the best picture of bone changes correlated to the distobuccal movement of the teeth (Figs. 6 and 7). The picture on the mesial alveolar wall and in the mesial area of the periodontal membrane is fairly uniform. The apposition of bone and the presence of strong, regular, taut principal fibers in the periodontal membrane are characteristic for this area (Figs. 8, 9, and 10).

The bone forming the distal alveolar wall and the distal part of the periodontal membrane reveals striking differences in different specimens. A cycle of three consecutive stages in this area can be observed. In the first stage the bone shows all the signs of active osteoclastic resorption. The periodontal membrane has lost the functional arrangement of the principal fibers almost entirely and consists of irregular, dense connective tissue (Fig. 8). In the second stage thin layers of bone are deposited on the resorbed surface in isolated areas. Thus, principal fibers of the periodontal membrane are reanchored to the distal alveolar wall and the periodontal membrane has gained a more regular structure (Fig. 9). In the third stage the reparative apposition

of bone has advanced farther and the difference between mesial and distal parts of the periodontal membrane has almost disappeared (Fig. 10).

These observations prove that the distal drift, although lifelong, is intermittent. During the period of rest, resorption of bone is repaired to an extent just sufficient to allow the reattachment of periodontal fibers and the restoration of normal function.

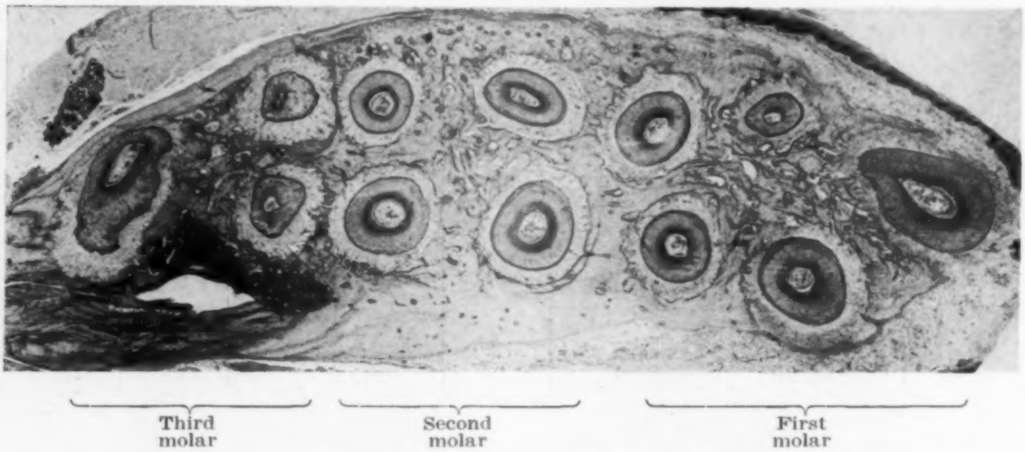


Fig. 6.—Horizontal section through maxillary molars.

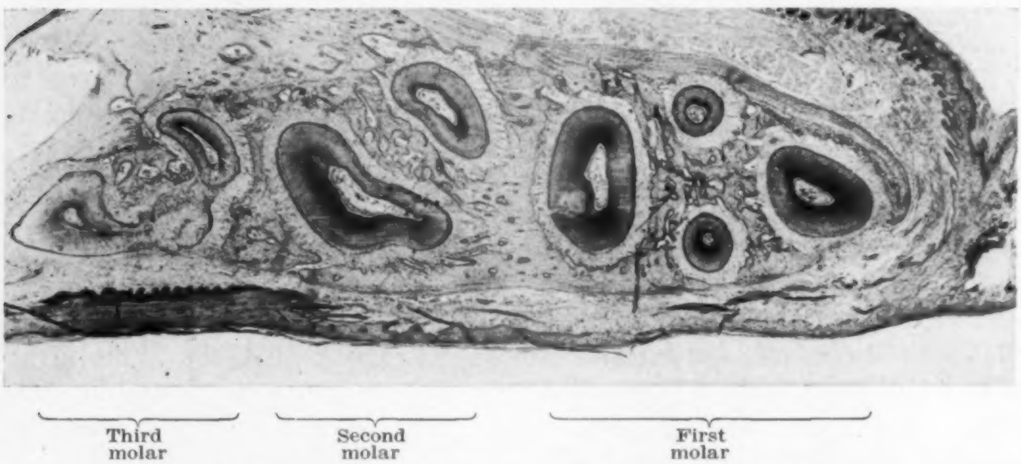


Fig. 7.—Horizontal section through mandibular molars.

Often the distal surfaces of the roots show shallow resorptions. In many of these areas, more or less complete repair has occurred by apposition of cementum on the resorbed surface of cementum or dentine (Figs. 8 and 9).

Ground sections, through the jaws of rats injected with repeated doses of alizarin-S, show the apposition of bone proceeding at the fundus, at the crests of the interdental and interradicular septa, and on the distal surfaces of the septa (Fig. 11). Alizarin injections permit an estimate of the weekly apposition of bone associated with the distal drift of the teeth and, therefore, the extent of this movement. It could be established that the upper and lower molars of young rats drift distally at a rate of about 60 to 80 microns a week.

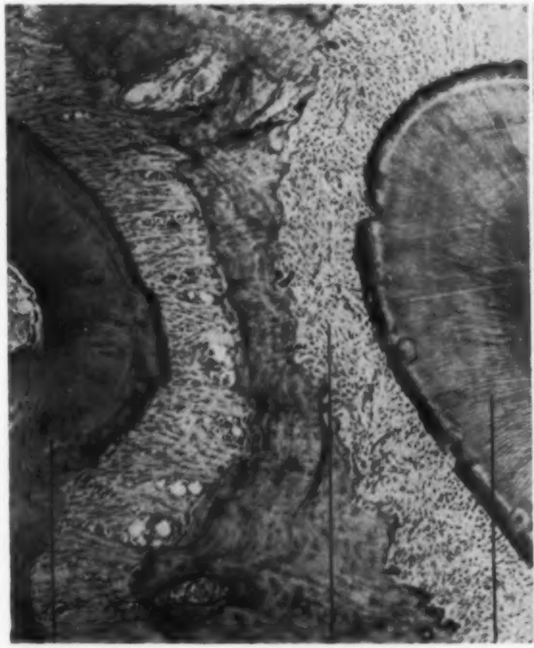


Fig. 8.—Horizontal section through the septum between mandibular second and third molars. Disorganization of periodontal membrane.

Fig. 9.—Horizontal section through mandibular first and second molars. Beginning reorganization of periodontal membrane.

Fig. 10.—Horizontal section through the septum between the lingual roots of maxillary second molar. Complete reorganization of periodontal membrane.

MOVEMENT OF THE INCISORS

The analysis of the changes in the incisal region is far more complicated than in the molar region because of the following factors:

1. The incisors grow continuously and erupt continuously by their longitudinal growth. The basal end of the incisors is a fixed plane during eruption. This plane is indicated histologically by the epithelial diaphragm which rests upon the hammock ligament.[14]

2. As mentioned before, the incisor increases in actual size during the whole life of the animal. This growth is proportionate to the growth of the whole animal and more specifically to the growth of the jaws.

Fig. 11.—Sagittal ground section through mandibular molars of a rat which had received three alizarin-S injections. Bone apposition at the alveolar crests and at the distal surfaces of the alveolar septa.

3. Concomitant with the increase in size, the incisors also change their shape, gradually flattening their curvature. Consecutive stages form segments of a continuously widening spiral.[8]

The complexity of the changes makes it advisable to study the movement of the incisors first in roentgenograms. Thus, a gross concept of their movement can be gained to be then compared to the histologic findings in the alveolar bone.

The Lower Incisor.—A study of roentgenograms of lower jaws of different ages shows that the mesiodistal relation between incisor and molars remains fairly constant in spite of the continuous distal drift of the molars (Fig. 12). This indicates a distal shift of the incisor. At the same time the vertical relation between the *fundus* of the incisor and the molars remains fairly constant in spite of the continuous vertical eruption of the molars. This relation

can be maintained only by an upward movement of the basal end of the incisor. Molars as well as incisors, moving distally, move at the same time buccally, following the divergence of the two mandibles.

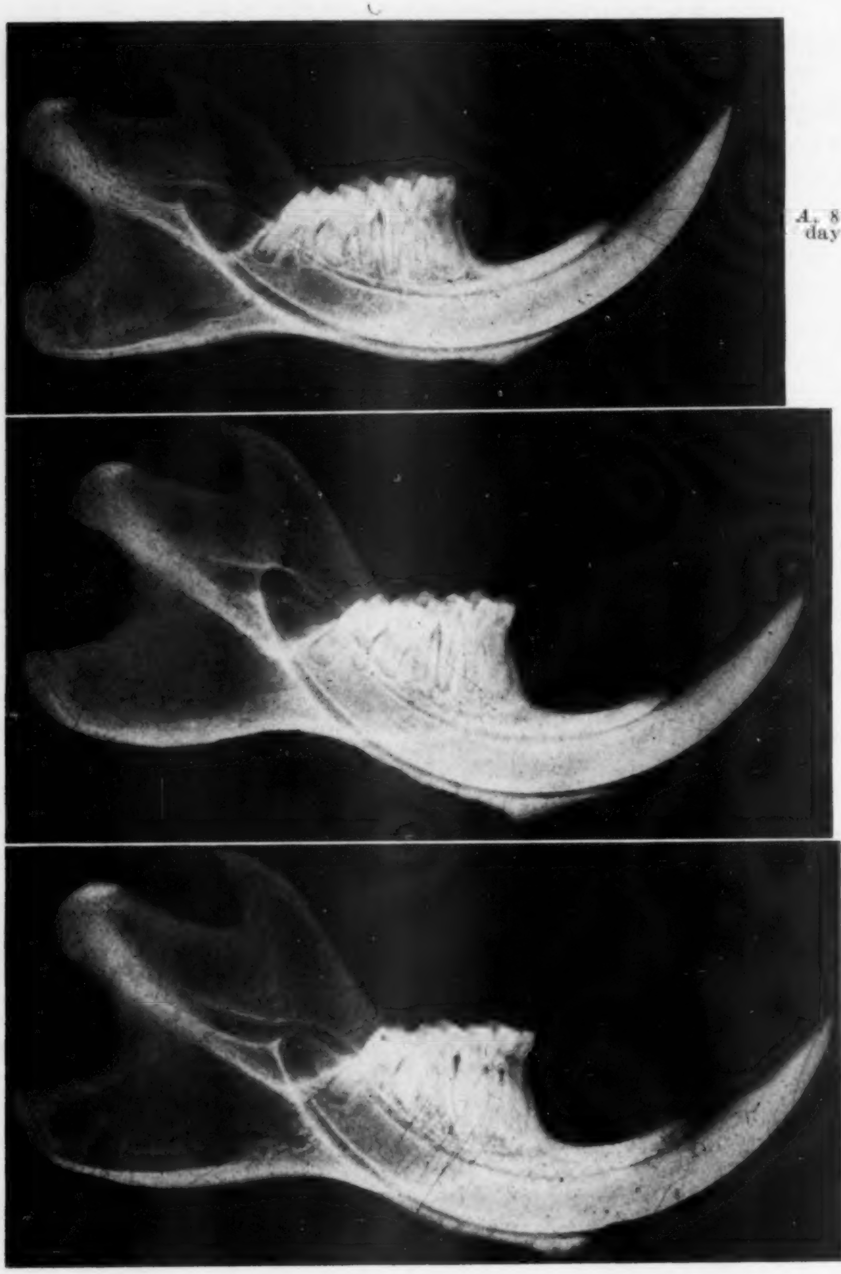
The superposition of roentgenograms of lower jaws of rats of different ages gives a clear picture of the peculiar movement of the incisor (Fig. 13). The superposition has to be done in such a way that the lower borders of the mandibles are made to coincide, because there is no appreciable apposition of bone at the lower mandibular border after the second week of postnatal life. If the known extent of the occlusodistal movement of the molars is taken into account, then the *mesiodistal* relations of the two jaws can be established, and it can be shown that the following anatomical landmarks coincide:

1. The posterior ventral end of the mandibular symphysis, which appears in roentgenograms as a sharp triangular spine at the lower border of the mandible slightly anterior to the first molar.
2. The crista colli mandibulae.
3. Sometimes the anterior borders of the coronoid process.

The study of two superimposed roentgenograms (Fig. 13) shows the enlargement of the incisor and the straightening of its curve. Most important is the fact that the fundic bone remains in the same plane in spite of the extension of the fundus upwards and backwards corresponding to the occlusodistal movement of the basal end of the incisor. The plane of the fundic bone is parallel to the plane of the crista colli mandibulae and, therefore, parallel to the plane of condylar growth. Although the incisor seems to grow into the mandible, the plane of the fundic bone, the epithelial diaphragm, and the hammock ligament remains fixed: *The basal end of the incisor glides along this fixed oblique plane.* Thus, the basal end of the incisor remains stable in relation to the long axis of the tooth. The hammock ligament, though growing in diameter, remains the fixed structure on which the tooth rests and from which it grows in length and thus erupts.

Corresponding to the lack of bone apposition at the lower border of the mandible, the convex surface of the lower incisor in the middle one-third of the tooth remains in a relatively fixed position. Therefore, the thickening and flattening of the incisor widens the alveolus in the middle one-third of the tooth at its concavity. This can also be proved by the comparison between rate of eruption of the molars and the increase in the distance between root apices of the first molars and concavity of the incisor.[9] The increase in the distance between molars and incisor is far less than the distance the molars move occlusally during their continuous eruption. In the basal one-third of the incisor, the widening occurs at the convexity of the tooth. The incisal one-third is, for a great part, extra-alveolar and does not show significant changes of the bone on the roentgenograms.

The histologic findings in the alveolar bone of the lower rat incisor correspond in every detail with the findings gained from the roentgenograms. The thin plate of the fundic bone is in an aplastic state; neither apposition nor resorption can be observed. The periodontal surface of the alveolar bone

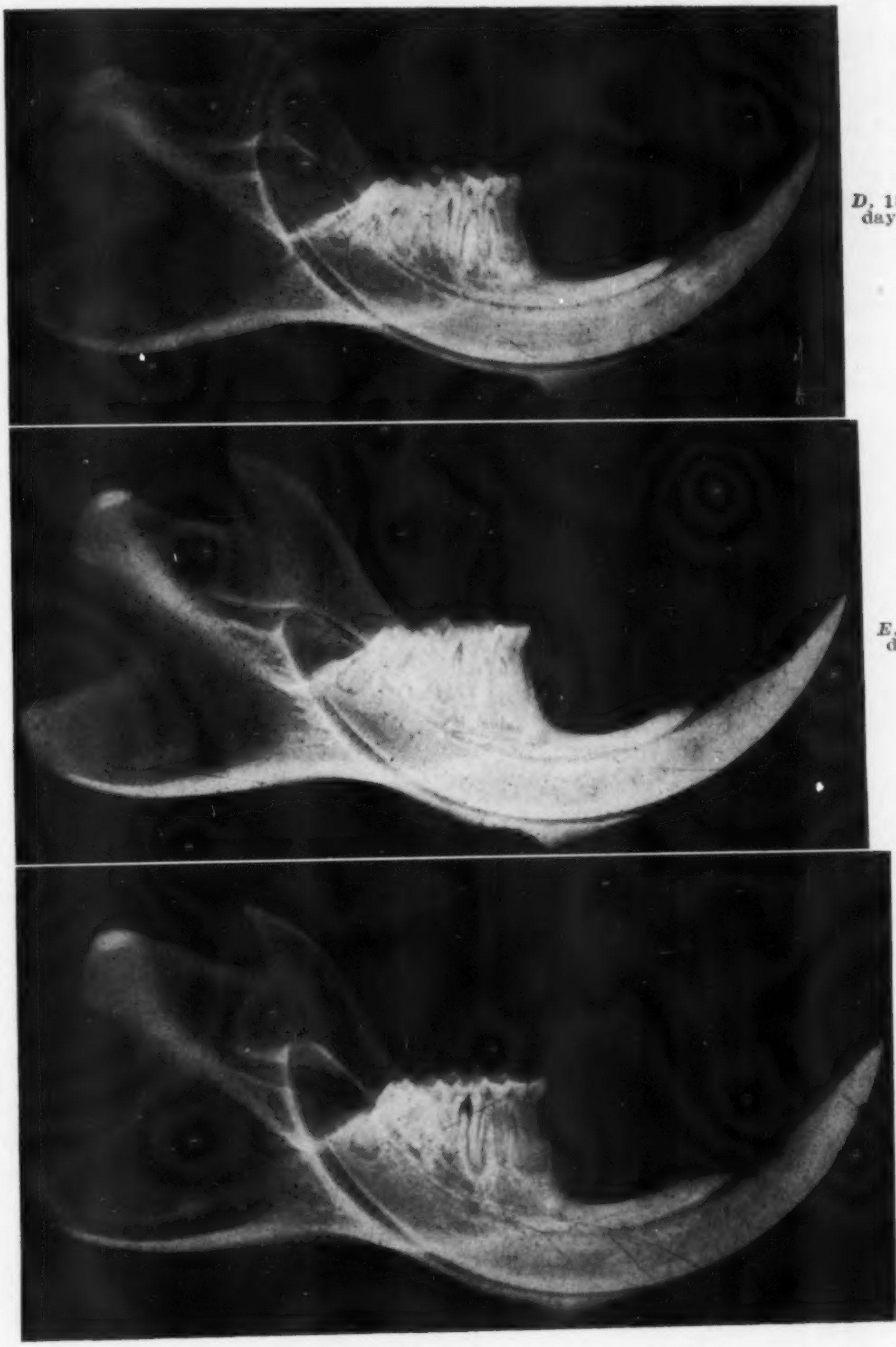


A, 84 days

B, 114 days

C, 153 days

Fig. 12.—Roentgenograms of rat mandibles of six consecutive growth stages. Relations between teeth and jaw and between incisor and molars are constant.



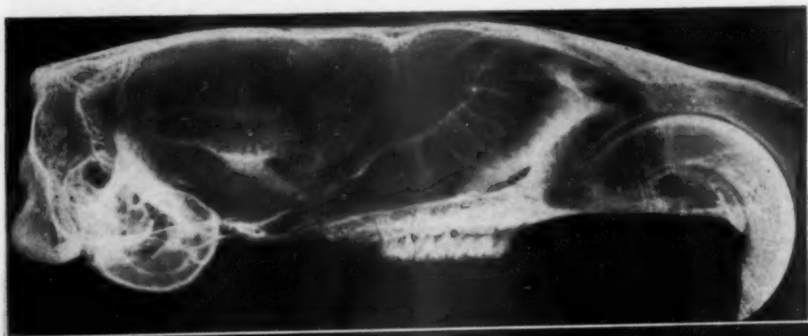
D, 187 days

E, 212 days

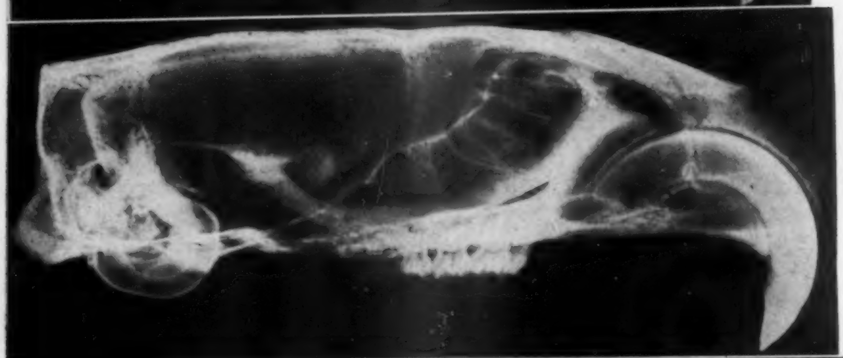
F, about 300 days

Fig. 12. (*Cont'd*).—*See opposite page for legend.*

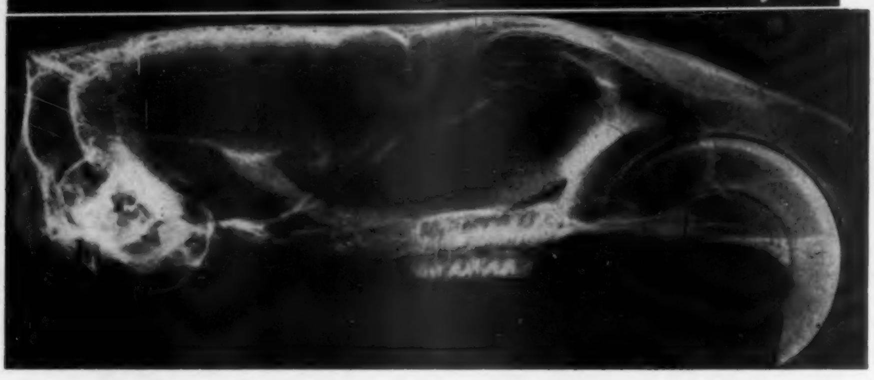
Fig. 13.—Superimposed outlines of the roentgenograms of animals *B* and *E* in Fig. 12. **Broken line**: animal 114 days old. Solid line: animal 212 days old. Growth areas of mandible; occlusodistal movement of molars; growth and shift of incisor.



A, 84 days



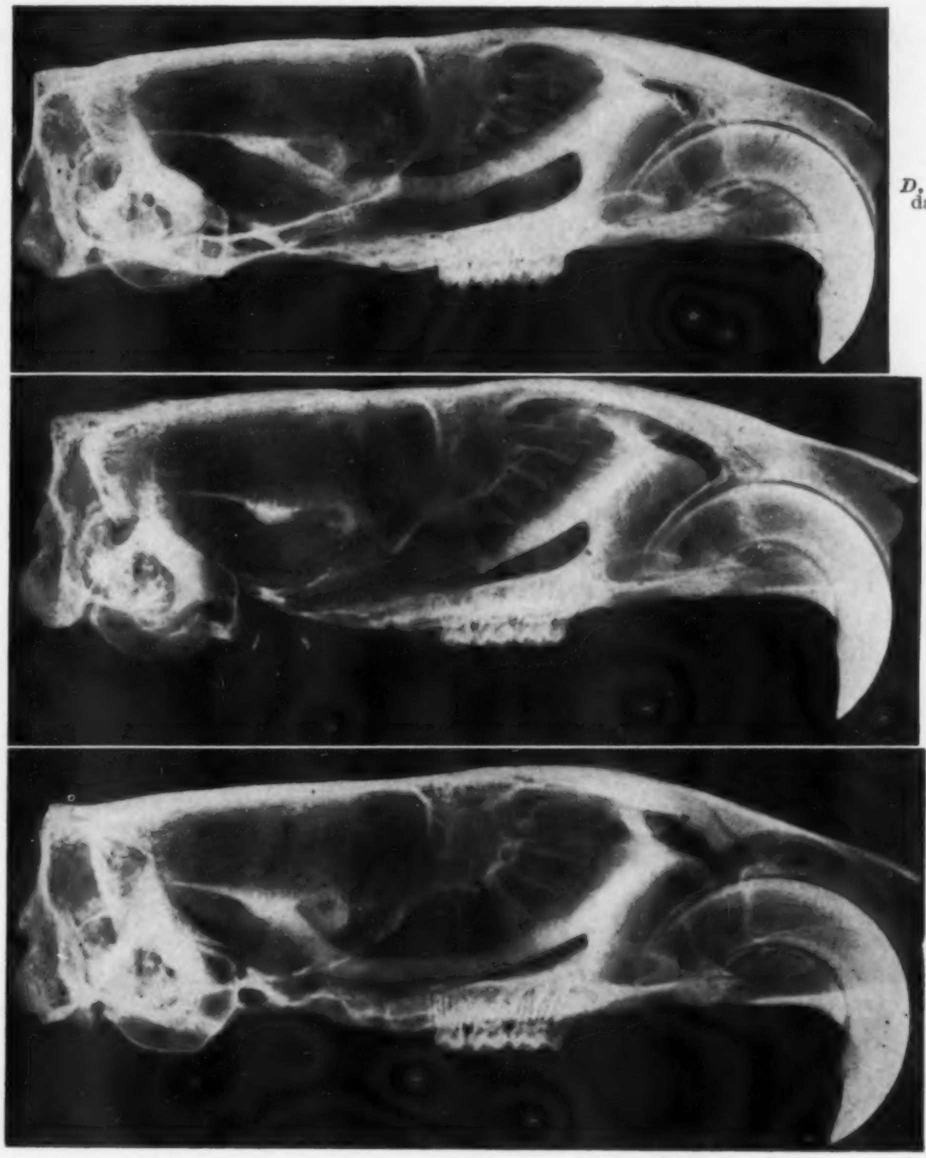
B, 114 days



C, 153 days

Fig. 14.—Roentgenograms of rat skulls of six consecutive growth stages. Relations between teeth and jaw and between incisor and molars are constant.

opposite the enamel-covered convex side of the tooth shows active resorption in the basal half of the alveolus, corresponding to the basal one-third of the tooth. The resorption is most active at the junction with the fundic bone and diminishes gradually in the incisal direction. The bone in the mesial half of the alveolus, corresponding to the middle one-third of the tooth, is relatively at rest. Osteoclastic giant cells as well as osteoblasts are absent in this area.



D, 187 days

E, 212 days

F, about 300 days

Fig. 14 (*Cont'd*).—*See opposite page for legend.*

The concavity of the tooth is covered by cementum and anchored to the bone. Apposition of new bone occurs in the region close to the fundus. In the area corresponding approximately to the area of the first molar, the bone shows all the signs of resorption. The incisal part of the alveolar bone at the concavity corresponding to the diastema between the incisor and molars shows active apposition at the periodontal surface, increasing toward the free margin.

The Upper Incisor.—Comparison of roentgenograms of the skulls and upper jaws of rats of different ages shows that the basal end of the incisor maintains a constant relation to the molars (Fig. 14). Since the molars move occlusodistally, the basal end of the incisor must shift in the same direction. The direction of the movement is indicated by the inclination of the fundic bone, which slopes backward and slightly downward.

Superposition of roentgenograms shows that the thickening of the incisor and widening of its curvature occur in an almost concentric manner (Fig. 15).

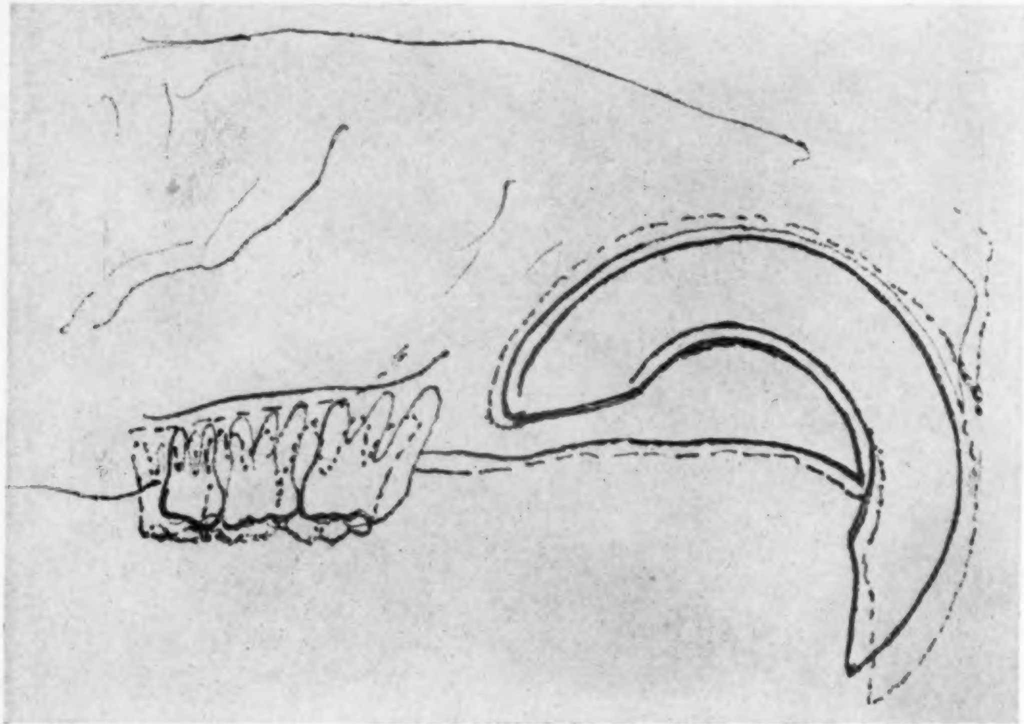


Fig. 15.—Superimposed outlines of roentgenograms of animals B and D in Fig. 14. Solid line: animal 114 days old. Broken line: animal 187 days old. Occlusodistal movement of molars; growth and shift of incisor.

The histologic findings on the alveolar bone of the upper rat incisor are interestingly complicated by the division of the alveolar bone into two parts: one formed by the premaxilla, the other by the maxilla. Although this fact is well known, it has never been considered in biological and experimental studies on rats. Many observations become of value only if the relations of the premaxillo-maxillary suture to the upper incisor are well understood. In central longitudinal sections the suture is found on the concave side of the tooth passing through the alveolar bone very close to the fundus (Fig. 16). On the convex side the suture is slightly anterior to the plane in which it is found on the concave. The maxilla forms, therefore, an irregular cup-shaped basal part of the alveolus. It covers, on the convex side, approximately the basal one-third of the tooth, forms the fundic bone, and reaches just to the lingual surface of the tooth. The incisor is almost entirely anchored to the premaxilla, which forms most of the alveolar wall on the concave surface.

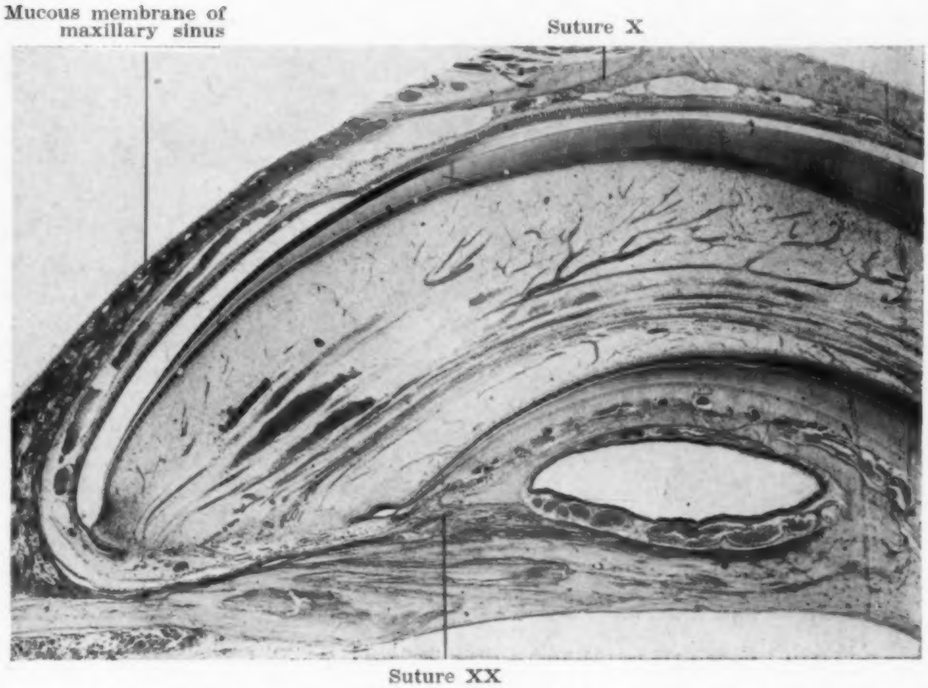


Fig. 16*A*.—Central section of basal half of maxillary incisor. Relation of premaxillo-maxillary suture to the tooth.

Fig. 16*B*.—Diagram of the alveolus of maxillary incisor. Maxilla stippled, premaxilla crosshatched. The sites of bone apposition indicated by + signs; the sites of bone resorption indicated by – signs.

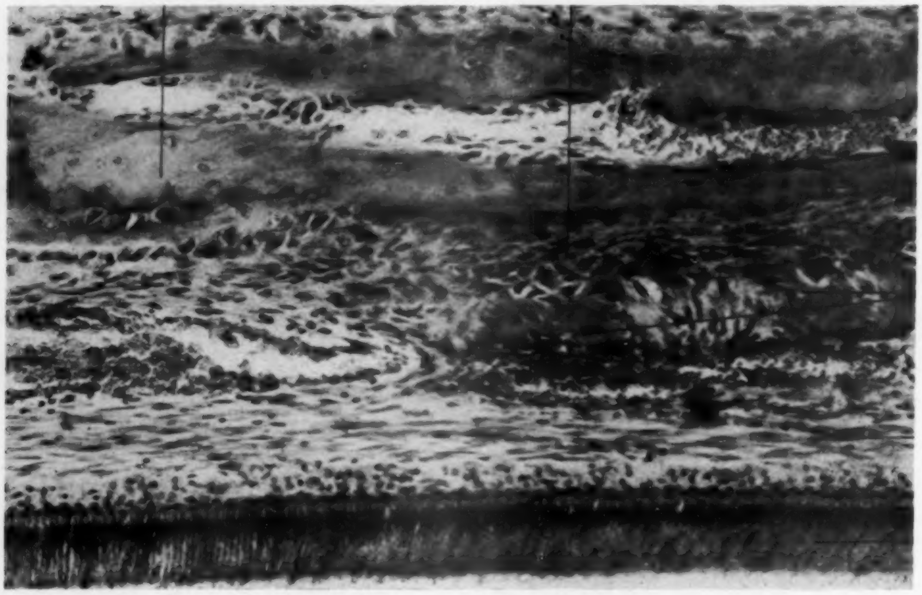


Fig. 17.—Periodontal surface of maxillary incisor alveolus at the convex side of the tooth. Bone apposition on the maxilla, bone resorption of the premaxilla. Area *X* of Fig. 16*A*.

Fig. 18.—Periodontal surface of maxillary incisor alveolus at the concave side of the tooth. Bone apposition on the premaxilla, bone resorption on the maxilla. Area *XX* of Fig. 16*A*.

As far as the alveolus is formed by the premaxilla, the changes on the periodontal side of the alveolar bone correspond to the movement of the tooth seen in the roentgenograms. The entire alveolar bone on the concave side shows apposition. This is most active in the incisal and basal regions, least active in the intermediate area. The alveolar bone on the convex side, anterior to the premaxillo-maxillary suture, shows active resorption.

The findings in the maxillary part of the alveolar bone are paradoxical. The fundic bone, which, according to the roentgenograms, is a fixed plane, shows active resorption. The small part of the alveolar bone at the basal end on the concave side shows resorption although the entire premaxillary part of the alveolar bone opposite the concavity of the tooth shows apposition (Fig. 18). The bone opposite the basal third of the convexity of the tooth shows active apposition, whereas the premaxillary part of the alveolar bone opposite the convexity is continuously being resorbed (Fig. 17).

The explanation for these findings is to be found in the presence of the premaxillo-maxillary suture and the appositional growth of bone in the suture. The sutural growth expands and distorts the alveolus. The plane of the suture being almost vertical, growth in the suture will shift the maxillary bone in such a way that the oblique fundic bone and the adjacent part of the aveolar bone on the concave side of the tooth are moved toward the tooth itself. The maxillary part of the alveolar bone opposite the convexity of the tooth is moved away from the enamel-covered surface of the tooth.* The resorption of bone at the fundus is a simple reaction to this movement to maintain the width of the apical space. The apposition of the bone along the convexity of the tooth, however, can be understood only if we recognize that the sutural growth leads to a widening of the periodontal space in the maxilla beyond the widening necessary to accommodate the enlarging tooth. The widening of the periodontal space by the sutural growth provides space not only for the growth of the tooth but also for the thickening of the bony lamella separating alveolus from maxillary sinus. The thickening of this bone during the growth of the skull is thus possible without encroachment on the maxillary sinus. The maxillary part of the alveolar bone on the concave side is moved toward the tooth faster than to compensate for the widening of the curvature of the incisor. Instead of apposition which is found at the concavity of the incisor in the premaxilla, the small maxillary part shows resorption on the periodontal surface.

DISCUSSION

In man, the balance between dentition and growing jaws is safeguarded by two main factors. First, the replacement of the deciduous teeth by their larger permanent successors and, second, the successive eruption of the posterior teeth into the lengthening jaws. The development of the teeth and their eruption extend over almost the entire growth period of the jaws. During this time, the vertical eruption of the teeth, after they have reached the occlusal plane, is correlated to the vertical growth of the jaws, which occurs mainly at the free alveolar borders. The continuous vertical eruption of the

*In reality the movement is probably reversed; i.e., the tooth is moved toward the fundic bone and away from the bone at the convexity. For an explanation of the bone changes, however, only the relative change in position is of importance.

human teeth in later life, along with their slight lengthening by apposition of cementum at the root apices, is primarily a compensation for the loss of tooth substance by occlusal attrition. The simultaneous mesial drift is a compensation for the loss of tooth substance at the contact point from interproximal abrasion.

The rat has only one generation of teeth corresponding to the permanent dentition of other mammals. The molars erupt in the second to fifth week of postnatal life in a position which is in balance with the attachment of the muscles of mastication. During the growth of the jaws, which extends over the entire life of the animal, this balanced position tends to be continuously disturbed. The main reason for this is the fact that the jaws grow mostly at their posterior ends. The distal drift of the molars is the compensation for these growth changes of the jaws.

The rate of distal drift is the same in the upper and the lower jaw, although the upper jaw grows not only in the pterygomaxillary suture at its posterior end, but also in the premaxillo-maxillary suture between molars and incisor. The reason for this paradoxical behavior is the fact that the upper jaw is relatively much longer than the lower jaw; its absolute increments are, therefore, greater. The premaxillo-maxillary suture provides this additional growth since the growth at the posterior end of the maxilla is equal to the total longitudinal increment of growth in the mandible.

The continuous vertical eruption of the molars and their elongation by cementum apposition is, in the rat, a consequence of the continuous growth of the jaws in height and a compensation for the regularly advancing attrition.

The molars of the rat are teeth with limited growth. No enlargement of their occlusal surface is possible and thus no adjustment of their size to the growing masticatory apparatus. The growth of the rat molars by cementum apposition on their roots plays, therefore, morphologically, a very inconspicuous role in the growth changes of the animal. The adaptive changes are almost entirely expressed by the continuous occluso-disto-buccal movement of the molars. This movement is associated with continuous growth changes of the aveolar bone, i.e., with precisely directed apposition and resorption.

Though morphologically inconspicuous, the continuous apposition of cementum plays a very important role *biologically* in the movements of the molars. The different behavior of bone and cementum toward pressure is well known.[6, 12] The fact that cementum resists resorption much more than bone is the reason that orthodontists can move teeth successfully. It is the peculiar growth potential of the cementum which explains its resistance to resorption. Normally, cementum grows by apposition to its entire surface throughout the life of an individual. A thin layer of cementoid tissue always covers the entire root, and, just as is the case with osteoid tissue or predentine, this uncalcified tissue resists osteoclastic resorption.[11]

Bone, on the other hand, is characterized by the presence of restricted and exactly defined apposition areas during the evolution of its growth pattern. In the rat, such areas of appositional growth are found, for example, on the distal surface of the aveolar septa, the alveolar fundus, and the alveolar crest. Apposition of bone on the mesial alveolar wall will produce a slight

increase in tissue pressure directed distally toward the root. The tooth surface being relatively immune to resorption, this pressure will be transformed into a distal *movement* of the tooth leading to an increase in pressure in the distal part of the periodontal tissue. This pressure causes resorption of the distal alveolar wall, and thus a recovery of the pressure balance.

Differential growth, expressed in the different growth potentials of cementum and bone is, therefore, the cause of physiologic tooth movement.*

The incisor of the rat is a continuously growing tooth, the basal area of which remains throughout life in an embryonic stage. The adaptation of this tooth to the growth of the animal and its jaws is effected, therefore, primarily by growth changes of the tooth itself. The first of these changes consists in continuous elongation of the tooth, not only compensating for the loss of tooth substance at the cutting edge but leading to an actual growth of the incisor in length. Then, the tooth gains in its transverse diameters, at the same time, so that its volume and weight remain approximately proportionate to the body weight of the animal. In addition, the incisor changes its shape continuously, the radius of its spiral constantly enlarging. Thus, a straightening of the incisor adapts the shape of the tooth to the lengthening jaw.

Although the growth changes of the tooth seem to govern the development of the incisor, changes in the surrounding alveolar bone also play a very important role. This can be seen clearly in the rat's lower incisor where the apposition of bone on the concave side of the tooth near the free alveolar border serves to stabilize the convexity of the tooth at the inferior mandibular border. Similar observations can be made on the upper incisor where the graded apposition of bone in the premaxilla opposite the concavity of the tooth maintains the proper relations between tooth and jaw. Bone apposition and consecutive bone resorption are, therefore, the guiding principles which keep the incisor, though ever changing in size and shape, in its correct anatomic and functional position. The cementum on the concave surface of the incisor plays the same important role as in the molars. By its inherent continuous growth potential, the cementum protects the tooth from resorption. On the convex surface of the incisor the enamel is protected against resorption by the epithelial enamel organ.

During all these changes of the incisors, a fixed plane is provided by the hammock ligament[14] as a structure upon which the basal end of the tooth rests, so that its longitudinal growth is transformed into continuous eruption. This plane remains fixed when the incisors move during the growth of the jaws by a directional apposition of bone; the basal end of the tooth glides along this oblique plane into the jaw. It is interesting to note that the angle of inclination of the basal plane is much greater in the lower jaw than in the upper. The explanation for this difference lies in the fact that the growth of the mandibular ramus in height is equal to the vertical growth of upper and lower alveolar processes. Therefore, the upward component of the movement of the lower incisor is greater than the downward component of the movement of the upper incisor.

*The term "differential growth" describes the fact that different organs or different parts of an organ grow at a different rate (and start to grow at different times).

The observations of the close interrelationship between movement and growth changes of the teeth and of the bone give conclusive proof for the statement that movement of the teeth is but a part of the general growth of the jaws, the skull, and the animal.

An experiment of Nature gives additional proof of the unity of changes in the dentition, whether they are expressed in changes of the size and shape of a tooth or its position. In the alveolus of the upper incisor, sutural growth between premaxilla and maxilla interferes directly with the relations between tooth and alveolar bone. The consecutive corrective changes in the walls of the maxillary part of the alveolar bone shed an interesting light upon the inseparable unity between tooth and bone changes.

We are forced to look upon tooth movement as an integral part of the elaboration of the growth pattern of the jaws and to consider growth of bone as the "force" which moves the teeth. A critical review of other theories on the dynamics of tooth movement is necessary to examine further the validity of this statement. In order to simplify the discussion, we will confine it to the movement of fully developed teeth. Two theories have been advanced as to the causes of tooth movement: the *vascular* theory and the *orthodontic* theory. The vascular theory maintains that the roots of teeth embedded in the jaw are under vascular pressure of the periodontal tissues; the crown reaching into the oral cavity is free from such pressure, and the tooth, therefore, is propelled into the oral cavity. If this theory were correct, the vertical eruption of a tooth could only be stopped by its coming into occlusion with its antagonist. However, comparative anatomy furnishes many examples to the contrary. One has only to remember the rudimentary canines of the male horse or the camel which never come into function or into occlusion. Besides, the vascular theory could only explain, at the very best, the vertical movement of teeth and not the bodily drift of teeth mesially or distally.

The orthodontic theory starts from the correct observation that, during orthodontic treatment, the tooth is actually moved, whereas the bone behaves as a passive tissue, giving way by resorption on the pressure side and being rebuilt by apposition on the side of tension. However, the conclusion drawn from these findings, that, also, during normal development bone can only be a passive tissue, is not permissible. In order to create an analogy between orthodontic and physiologic movement of teeth, the followers of the orthodontic theory have looked upon masticatory forces as the primary cause for the physiologic movement of teeth. This theory could explain, at the best, the drifting movement of teeth or their tilting. It is, however, not capable of explaining the continuous vertical eruption of fully developed teeth. Furthermore, the mesial drift of human teeth has been explained by the mesial inclination of upper and lower molars.[3] This position was said to provide the pressure directed mesially. In the rat, however, the lower molars are inclined mesially, whereas the upper molars are inclined distally. Still, both the upper and lower molars always drift distally.

The findings described in this study and the findings on developing and erupting teeth[14, 15, 17] seem to justify the following *general conclusion:* Developmental movements of tooth germs, eruptive movements of teeth, and physiologic movements of teeth during their functional period are the result of

differential growth of the tooth and the surrounding bone. In different periods (developmental, prefunctional, and functional period) and in different types of teeth (continuously growing teeth and teeth with limited growth), growth of tooth and growth of bone participate to a variable degree in affecting tooth movements. Movement of the teeth is, therefore, but a component of the growth of the jaws in the elaboration of the genetically determined growth pattern.

SUMMARY AND CONCLUSIONS

1. The physiologic movements of the teeth and the growth changes of the bone were investigated on the albino rat. An attempt was made to decide which of these associated phenomena is the primary. The studies were made by anatomic, roentgenographic and histologic methods. Periodic injections of alizarin-S were used to establish histologic landmarks.

2. A detailed description of the rat dentition and its relation to the skeleton was given. Special attention was given to the fact that premaxilla and maxilla participate in forming the alveolus of the rat's upper incisor.

3. It was found that the mandible grows in length at the condyle and at the posterior borders. The ramus grows in height at the condyle and the tip of the coronoid process, the body at the free borders of the alveolar process. The absolute growth of the maxilla in length is greater than that of the mandible. Pterygomaxillary and premaxillo-maxillary sutures contribute to the elongation of the maxilla. The body of the maxilla grows in height by apposition of bone at the alveolar border.

4. The molars erupt early in life in the growing jaws. Their continuous vertical eruption is not only compensatory for attrition but correlated to the growth of the jaws in height. The molars drift distally throughout life in correlation with the growth of maxilla and mandible in length at their posterior ends. Apposition of bone at the fundus and root elongation by apposition of cementum characterize vertical eruption. Apposition of bone on the mesial alveolar wall, resorption of bone at the distal wall, and continuous apposition of cementum on all root surfaces indicate the distal drift. This movement is intermittent, as shown by reparative apposition of bone on the resorbed surfaces and the alternating disorganization and reorganization of the periodontal membrane.

5. The incisors grow and erupt continuously. Their growth is not only compensatory for attrition but leads to an absolute increase of the incisor in all its dimensions. The plane of the basal end of the incisors remains fixed. The incisors grow along the oblique plane of the basal end into the jaws. The basal end of the lower incisor moves distally and superiorly, that of the upper incisor distally and inferiorly. The lower incisor follows, by its upward movement, the growth in height of the mandibular ramus; the upper incisor follows, by its downward movement, the growth in height of the maxillary body. The upward component of the movement of the lower incisor is far greater than that of the upper incisor because the vertical growth of the mandibular ramus equals the vertical growth of the body of both upper and lower jaws.

6. In the upper jaw where growth in the premaxillo-maxillary suture interferes directly with the relations between alveolar bone and tooth, bone

changes around the incisors are highly complicated. The relations between suture and tooth and the peculiar bone changes in the maxillary part of the alveolar bone are described.

7. Although the axial eruption of continuously growing teeth is primarily the expression of their longitudinal growth, bone apposition and resorption keep such teeth in the proper anatomical and functional position.

8. The close correlation between growth of the jaws, in one instance, and growth of the teeth and movements of the teeth in the other, leads to the conclusion that tooth movement is but one component of the general growth of the animal in the elaboration of the genetically determined growth pattern.

9. Growth of bone in circumscribed, exactly defined areas is the direct cause for tooth movement. The continuous growth potential of the cementum plays an important role in this process. The constant presence of a superficial layer of uncalcified cementoid renders the tooth relatively immune to resorption. Thus, the pressure caused by growth (apposition) of bone on one alveolar wall is transmitted by the moving tooth to the opposite alveolar wall where it causes resorption.

10. Movement of teeth is the expression of differential growth of tooth and bone.

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LUDWIG'S ANGINA

An Anatomic Study of the Role of the Lower Molar Teeth in Its Pathogenesis

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Dr. Wilhelm Friedrich von Ludwig[1] deserves a permanent place in medical history because he recognized the disease now called by his name as a distinct morbid entity, characterized by anatomic, pathologic, and clinical signs, peculiar to it and differentiating it from lesions closely resembling it. Of course, his assumption that he had met with a new kind of inflammation of the neck, of an epidemic character, was erroneous.

HISTORY

The disease had been known since classical antiquity and had been mentioned by Hippocrates, Galen, Caelius, Aretæus Paulus Aegineta, Aurelianus, Severinus, and others, and it appears in the later literature under various names. For example, it was classified as "cynanche," "carbunculus gangraenosus," "morbus strangulatorius," esquinancia gangraenosa," and "angina maligna." The Spaniards called it "garotillo" (after "garote," a loop used by hangmen) and the French "l'esquinancie inflammatoire gangreneuse" (Deberge, 1758) or "phlegmon large du cou" (Dupuytren, 1833). The occurrence of the disease had been observed and described by Colden in New York (1735) and by Cholmer (1770) in South Carolina. Dr. Thomas Kirkland (1786) reported a case of "angina externa" in which he saved the life of the patient by an early deep median incision, while General Washington succumbed to a hemorrhage caused by "cynanche trachealis."

A comprehensive history of the disease was presented by Parker,[2] in 1879, and by Muckleston,[3] in 1928. Many of the data had already been mentioned by Heim,[4] a contemporary of Ludwig, in questioning Ludwig's assumption that the disease was new.

Ludwig's Description.—These facts do not detract from Ludwig's merit. His classic article was a masterpiece of exact observation and precise description with an almost intuitive recognition of all the significant factors and characteristic details. "The short, clear, and comprehensive account which Ludwig gave of it has been surpassed by no later description of the disease" (Ashhurst[5]).

According to Ludwig's observations at the bedside and at the autopsy table, the observations to be emphasized as characteristic and the most important are:

1. Ludwig's angina is a rapidly spreading gangrenous cellulitis.
2. It originates in the region of the submaxillary gland (i.e., spatium submaxillare or submandibulare).

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From the Department of Otolaryngology, University of Cincinnati College of Medicine.

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3. The extension evidently occurs by continuity and not by the lymph channels.
4. It shows no special tendency to form abscesses.
5. There is plainly no involvement of the submaxillary glands and lymph nodes.

Dissenting Views.—However, despite Ludwig's exactness, some of those sharply defined characteristics "by which this disease differentiates itself from those other diseases whose outward appearance is somewhat akin" (Burke's[6] translation of Ludwig's article) have been frequently overlooked by various later observers. Roser[7] and others assumed that Ludwig's angina is a specific disease of the submaxillary salivary gland—a sort of submaxillary mumps. This idea, however, has been definitely abandoned.

Von Thaden,[8] Poulsen,[9] Stengel,[10] Thomas[11] and others considered the disease to be primarily a submaxillary lymphadenitis, even suggesting the term "submaxillary bubo" (von Thaden).

Furthermore, several French writers (Délorme, Leterrier, August, DeBovis and others) considered the disease as sublingual cellulitis. The term Ludwig's angina was even regarded as synonymous with sublingual phlegmon. There was a strong tendency to drop the term Ludwig's angina altogether, since the disease was not believed to deserve a special place in surgical pathology. This idea persisted intermittently down to the last decades (Finder,[12] 1925).

Pseudo Angina Ludovici.—At the beginning of the century, some useful work was done in order to restore the picture of the disease to its original distinctness. Davis[13] emphasized the fact that Ludwig's anginia spreads along connective tissues by direct continuity, is not transmitted by lymph channels, and does not effect lymph nodes. This was soon demonstrated histologically, by Coplin,[14] who was able to show that there is no infiltration of lymph nodes in Ludwig's angina.

On the other hand, Trautmann[15] mentioned a "secondary" type of phlegmon of the neck, which is actually caused by inflammation of lymph nodes consequent on infection in their drainage area. "This, of course, is not the same as the type described by Ludwig." A similar differentiation was made by Van Wagenen and Costello,[16] based on their careful studies and observations of sixteen cases. They even made a terminologic distinction between these two types using the terms "primary" and "secondary." Axhausen[17] and Wassmund[18] made a distinction between a phlegmonous and an "abscess-forming" type. They pointed to the fact that in the latter type the infectious agent passes by lymph channels throughout the submaxillary lymph nodes, whereas, in the phlegmonous type, it immediately spreads from the bone into the tissue of the floor of the mouth. None of these investigators observed any mortality with the so-called secondary or "abscess-forming" type.

It becomes clear that these diseases cannot be regarded as forms of Ludwig's angina. They actually are forms of suppurative submaxillary lymphadenitis enlarging in the direction of the mouth, a comparatively benign imitation of Ludwig's angina. In order to emphasize the outward similarity without identifying the disease with that described by Ludwig, I occasionally used in

discussion the term "pseudo angina Ludivici" or "pseudc Ludwig's" to designate this clinical entity.*

Dintinction from Sublingual Cellulitis.—Thomas[11] deserves the merit of having disapproved of the erroneous assumption advanced by the French school that Ludwig's angina is merely sublingual cellulitis. After a profound study of 104 cases described in the literature and two cases of his own, as well as anatomic studies, he was able to show that (1) Ludwig's angina starts almost regularly in the submaxillary space, (2) it only secondarily involves the sublingual space, and (3) the extension follows the route of the submaxillary salivary gland which turns around the posterior border of the mylohyoid muscle, passing through the gap in the buccopharyngeal wall. Isolated cellulitis either of the submaxillary space or of the sublingual one cannot be regarded as Ludwig's angina. *"It is the simultaneous involvement of both that warrants the condition being recognized as a distinct clinical entity"* (Ashhurst[5]).†

The importance of the mylohyoid muscle and its fascial layers as a factor determining the course and the character of the disease has been repeatedly pointed out (Piersol,[19] Houser,[20] Grodzinsky,[21] Williams,[22] and others). "It acts the same as a rubber band," said Novitzky,[23] "preventing escape of gas and fluid into the mouth." "These two structures (the mylohyoid muscle and the deep cervical fascia) therefore tend to confine infection following extractions of lower molars to the deep tissues under the tongue" (Trout[24]).

This muscle is also called "diaghragma oris" because it separates the neck from the floor of the mouth. Its upper surface belongs to the floor of the mouth; its lower surface belongs to the neck. Ludwig[6] described the disease as a "gangrenous induration of the connective tissues of the neck which advances to involve the tissues which cover the small muscles between the larynx and the floor of the mouth." In my opinion, therefore, the term "phlegmon of the floor of the mouth," which is frequently used as synonymous with Ludwig's angina, cannot be considered as entirely adequate to Ludwig's original description of the condition.

Etiology.—Ludwig's idea that the disease is epidemic has been frequently repeated in the last century but is now abandoned. The alleged occurrence of cases of Ludwig's angina in a greater number within a short time may probably be explained as the occurrence of secondary suppurative lymphadenitis in cases of a primarily epidemic disease such as scarlet fever, diphtheria, or measles. It is obvious that such infections can be regarded only as pseudo Ludwig's angina. Accordingly, those diseases frequently mentioned as sources of Ludwig's angina cannot be regarded as etiologically related to it. Likewise a number of lesions located in the drainage area of the submaxillary and submental lymph nodes have to be excluded. These are principally lesions about

*Hajek occasionally called it "Ludwigoid."

†These anatomic conditions differentiating Ludwig's angina from sublingual cellulitis are apparently of clinical and perhaps even of therapeutic importance. Since the sublingual space is separated from the oral cavity only by the overlying mucous membrane, an inflammatory process established merely in this space is more apt to empty into the mouth, either spontaneously or by an endoral incision (in contradistinction to a process extending below the diaphragma oris). Houser advocates the endoral incision on the basis of his successful results in several instances. This recommendation is rejected by others. Hauser's cases, however, were instances of isolated sublingual cellulitis as he emphasized. I believe the endoral approach may be justified as the shortest and simplest way to an area of sublingual cellulitis, but for submaxillary-sublingual cellulitis, which practically represents Ludwig's angina, the external approach should be the procedure of choice.

the lips, tongue, cheeks, gums, floor of the mouth, tonsils, and pharynx, and lesions of the skin such as impetigo, furuncles, erysipelas, lupus erythematosus, and otitis externa.

Since Ludwig's angina is originally a submaxillary cellulitis, it can be brought about, as a rule, by an infection which is primarily located below the mylohyoid muscle. The chief sources of infection mentioned in the literature are, in the approximate order of frequency of occurrence, an extraction of molar teeth (apparently the injection for anesthesia around a tooth is sometimes by contamination, a contributing factor), a periapical abscess, a penetrating injury of the floor of the mouth (gunshot wound, stab wound, entrance of a foreign body, horse kick, bite), osteomyelitis and compound fracture of the jaw, otitis media (by way of the digastric muscle, according to Piersol[19]), and an abscess under the thyrohyoid membrane (Blair[25]). Whether a salivary calculus, occasionally mentioned, is apt to produce Ludwig's angina seems to me doubtful. Poulsen,[9] discussing 251 cases of submaxillary inflammation, did not mention it as among the pathogenic factors as far as these have been revealed. The differentiation of an inflammatory swelling caused by an impacted salivary calculus from Ludwig's angina is of course sometimes difficult.* Moreover, peritonsillar abscess, although frequently mentioned, seems to me a rather questionable source of Ludwig's anginia because the palatoglossal muscle and partly also the styloglossal muscle form a protecting wall in the direction of the sublingual and submaxillary spaces. There may be confusions of peritonsillar abscess with periosteal alveolar abscess of the wisdom tooth as indicated by numerous writers (Trautmann[15]; Burchard[26]; Adrion[27]; Tschiassny[28]; Novitzky[29]).

The predominant role of the teeth among the pathogenic factors of Ludwig's angina was recognized comparatively late. Roser[7] rejected with remarkable vehemence this assumption when it was presented by Bamberger and Niemayer. Parker[2] mentioned periostitis of the jaw among the various etiologic factors but seemed to give it least emphasis. However, at the meeting of the French Surgical Society in Paris in 1892,[5] this condition was extensively discussed, and it has been proved to occur by numerous reports since that time. Ashhurst[5] estimated the frequency of a dental etiologic factor at 65 to 83 per cent; Trout,[24] at 82 per cent; and Wassmund,[18] at 90 per cent. Schlesinger[30] mentioned carious teeth as the only cause he was able to demonstrate, and Novitzky's[29] observations confirmed this. Reports emphasizing the high proportion of cases with a dental etiologic factor have been presented by Bell,[31] Muckleston,[3] Frankenthal,[32] Houser,[20] Ross,[33] Grodzinsky,[21] Price,[34] Taffel and Harvey,[35] and many others.

Which teeth are especially involved? Thomas,[11] reviewing the literature down to 1908, made the statement: "So far as reference to special teeth is concerned, they were always molar or wisdom teeth. . . ." Van Wagenen and Costello,[16] having reviewed the literature published since Thomas' review, 1928, reported the same observation. According to Wassmund, Richard and others, the back molar teeth are the most frequently involved. But several writers

*Several years ago I mentioned (in discussion on Wenzel, M.: Ein bemerkenswerter Fall von Speichelstein, Monatschr. f. Ohrenh. **70**: 747, 1936) a sign which I believe to be useful for this differential diagnosis. External pressure on the submaxillary region in a case of impacted calculus is followed by a cloudy discharge from the sublingual caruncle. This does not occur, of course, when there is nothing producing a stricture of Wharton's duct.

(Frankenthal,[32] Muckleston,[3] Grodzinsky,[21] Trout,[24] and others) have mentioned the involvement of the premolar teeth also. I was able to find only one report of Ludwig's angina caused by an extraction of a second bicuspid tooth as the only tooth involved. In this case, however, as emphasized by Frankenthal,[32] the dentist was faced with unusual difficulties which had necessitated the trephining of the alveolar process. Trautmann's[15] case was one of sublingual cellulitis following periapical abscess of a first premolar tooth. It is not regarded as a case of genuine Ludwig's angina.

Therefore, one is justified in assuming that (according to the observations of numerous writers throughout six decades) the dental focus of Ludwig's angina as far as it has been traced is located almost without exception about the molar teeth. The greatest predilection is shown for the second and third molar teeth, according to some observers.

An explanation of the etiologic predilection for the molar teeth was presented by Moty[36] at the meeting in Paris (1892): From a periapical inflammation following gangrenous pulpitis the pus pierces the alveolus in its thinnest part, which is on the outer surface for the anterior teeth. It forms in this way a dentoalveolar abscess. If the process is acute (as after ill-timed filling of a tooth), there is no time for absorption of the involved alveolus to occur. So the infection enters the dental canal, spreads toward to the spine of Spix and makes its way toward the cellular tissue which separates the upper surface of the mylohyoid muscle from the gum. If a back tooth or a wisdom tooth is infected in this way, the outer wall of the mandible is so thick that perforation occurs only on the inner wall, so that the sublingual tissues are infected at once.

This explanation of Moty has been frequently repeated in the literature with some unessential variations up to the present time and is apparently generally accepted (Piersol,[19] Novitzky,[29] Grodzinsky,[21] McCaskey,[37] and others). Is it conclusive? There is room for question whether the explanation given by Moty and his followers can be accepted as the indubitable and complete solution of the problem for the following reasons: (1) According to Moty's own words, "the infection . . . spreads toward the cellular tissue which separates the *upper* surface of the mylohyoid muscle from the gum . . . so that the *sublingual* tissues are infected at once." It therefore does not explain the infection of the submaxillary space which is, in fact, the primarily infected region, as has been proved by Thomas.[11]

(2) The anatomic conditions on which the explanation of Moty and his followers is based are, in fact, not quite as they are supposed to be by the authors. Transverse dissections of mandibles through the first, second, and third molar teeth, and the corresponding alveolus as performed by myself show that there is rather an irregularity in the differences of the thickness of the inner wall of the alveolus compared with the outer wall. Only as far as the third molar is concerned do the conditions confirm the statements of Moty. As for the first and second, sometimes there is no marked difference at all, and sometimes the proportions are just reversed, the inner wall being even thicker than the outer one. An example of that is presented in Fig. 1, *A*, *B*, and *C*.

Almost the same situation appears in illustrations of a paper by McMillan,[38] which, however, deals with the structures of the alveolar processes from a different point of view.

But even if one intends to assume that the situation in all instances of Ludwig's angina is of the type supposed by Moty, that would explain only why the molar teeth are more apt to transmit an infection toward the lingual side of the alveolus. But the question why just the submaxillary space should become infected in this way by the molar teeth and, morever, exclusively by the molar teeth, remains open.

Several other explanations presented by various authors cannot be accepted, I think, as an exact and entirely satisfactory answer. Thomas[11] simply remarked, in connection with the statement that the molar teeth are the only ones concerned, that they are nearest to the submaxillary region. Somewhat similar but more precise is the explanation given by Trout.[24] Emphasizing that the sockets of the molar teeth are "off center and closer to the inner than to the outer side," he said: "In addition to this anatomic fact it is important to recall that the molar teeth are separated from the underlying fossa of the lower mandible only by a relatively thin partition of the bone." Wassmund[18] expressed the belief that the proximity of the back molar teeth to the posterior border of the mylohyoid muscle makes it probably easier for an infection starting from those teeth to spread simultaneously into both the sublingual and the submaxillary space.

Fig. 1.—Cross sections through the mandible: *A*, first molar tooth; *B*, second molar tooth; *C*, third molar tooth. They demonstrate that the common statement that the lingual plate of the alveolar sockets of the molar teeth is thinner than the buccal plate is not invariably true. Note the difference in the relation of the mylohyoid ridge (*R*) to the apexes of the roots.

I should like to offer a report on the results of some anatomic studies which I undertook in order to find a solution of this problem. They are probably also of some interest because they reveal an anatomic detail which, as far as I know, has not been noticed before. They were based on the following considerations:

1. Ludwig's angina starts regularly from a focus below the mylohyoid muscle.
2. This muscle is attached to the mandible at the mylohyoid ridge.

3. Is there a special relation of the roots of the molar teeth to this ridge?

Textbooks of anatomy consulted did not offer any information in this connection. I, therefore, made examinations as follows: A narrow strip of lead covering the mylohyoid lines was fixed to the jaw on each side. Then roentgenograms were taken of each half separately. Three samples are shown in Fig. 2. Twenty-three jaws were examined in this way. In the majority of them, of course, many teeth were absent, one specimen even being unilaterally edentulous. Some teeth were markedly pushed upward by alveolar atrophic changes. Altogether, as far as concerns the molar teeth, there were present 27 first molar teeth, 32 second molar teeth, and 30 third molar teeth.

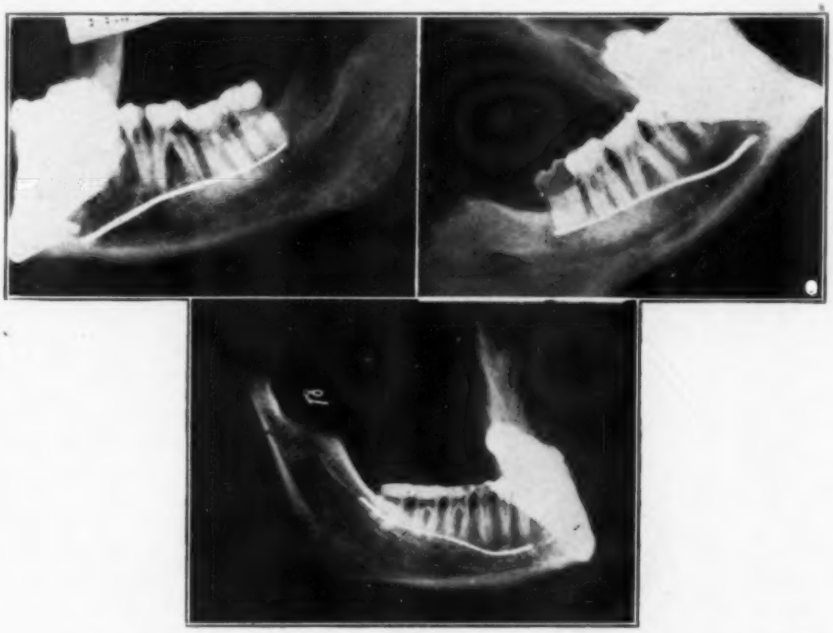


Fig. 2.—Roentgenograms showing the relation of the tips of the roots to the mylohyoid ridge.

The relation of the tips of the roots to the mylohyoid ridge, as the roentgenograms made plain, were as shown in Table I. It may be seen in the table that, with few exceptions, the tips of the roots of the second and third molar teeth reach below, or at least as far as the level of, the mylohyoid ridge, while a majority of the apexes of the first molar teeth are located above this ridge. As far as the anterior teeth are concerned, their root tips were without exception high above the mylohyoid ridge.

Table I

Relation of Roots of Molars to Mylohyoid Ridge

Molar	Teeth	No. with apex above the mylohyoid ridge	No. with apex about at the level of the mylohyoid ridge	No. with apex markedly below the mylohyoid ridge
I	27	16	8	3
II	32	5	6	21
III	30	3	8	19

In order to prove that there are no errors in the interpretation of the roentgenograms caused by faulty projections I performed the dissection shown in Fig. 3. A gap had been cut in the lingual plate of the mandible. The upper border of the gap represents approximately the level of the mylohyoid ridge. Fig. 3 shows the roots of the second and third molar teeth below the mylohyoid ridge. The tips of the first molar tooth are just visible at the level of the ridge. No roots of anterior teeth are visible.

These findings and figures show that under certain circumstances there is a great potentiality for the second and third molar teeth to become the source of submaxillary cellulitis since their roots reach most frequently below, or at least to the level of, the mylohyoid ridge. The first molar teeth, in contrast with this, are more inclined to transmit an infection into the sublingual space.

Fig. 3.—A groove on the inner side of the mandible shows the relation of the roots of the teeth to the mylohyoid ridge.

The proportion of the respective teeth related to the submaxillary space would be 11 (18 per cent) of the first, and 27 (41 per cent) of the second and of the third molar teeth.

These anatomic data further make it plain that occasionally an infected molar tooth or socket not reaching the level of the submaxillary space can become the source of sublingual cellulitis (Houser).[20]

It becomes clear also that the short distance between the border of the alveolar process and the mylohyoid ridge (about 4 to 10 mm. for the second and third molar teeth) renders it more likely that infectious agents will pass by way of an injection for anesthesia into the submaxillary space.[39]

Comparison With Proportions Compiled in the Literature.—It seemed to me of interest to investigate whether there was a correlation between these anatomic

findings and the frequency with which the three groups of molar teeth were etiologically involved in the cases which have been reported. Unfortunately, most of the available literature gives insufficient data by which to determine exactly the particular tooth involved as the primary focus of the infection. For the most part, one finds only such remarks as "A molar tooth was extracted" or "Caries of several lower molar teeth was noted." I was able to collect only 24 cases in which the teeth involved were determined. In six of them, however, two molar teeth were mentioned. Since it was not possible to determine which of the two was the causative one, I gave both of them the full credit in Table II.

TABLE II

INCIDENCE OF PRIMARY INVOLVEMENT OF EACH OF THE THREE MOLAR TEETH

AUTHOR	CASES	NUMBER IN WHICH INFECTION WAS REFERRED TO GIVEN MOLAR TOOTH		
		I	II	III
Ashhurst[5]	1	--	--	1
Blessinghame, C. D.: Tr. Am. Laryng., Rhin. & Otol. Soc. **34:** 33, 1928	2	1	1	
Conway, J. F.: J. A. M. A. **118:** 953, 1942	1	--	1	
Davis[13]	2	--	1	2
Grodzinsky[21]	3	1	1	3
Hochbaum, W. J.: Laryngoscope **43:** 838, 1933	1	--	--	1
Houser[20]*	1	--	1	
Medicolegal case[38]	1	--	--	1
Muckleston[3]	1	1		
Pratt, G. N.: Illinois M. J. **14:** 462, 1908	1	--	1	
Price[34]	2	1	1	
Ross[33]	2	--	1	2
Thomas[11]	1	--	1	
Van Wagenen and Costello[16]	4	2	2	2
Richard[40]	1	--	1	
Total	24	6	12	12

*Of course, it is not quite certain whether this case is one of Ludwig's angina or one of sublingual cellulitis.

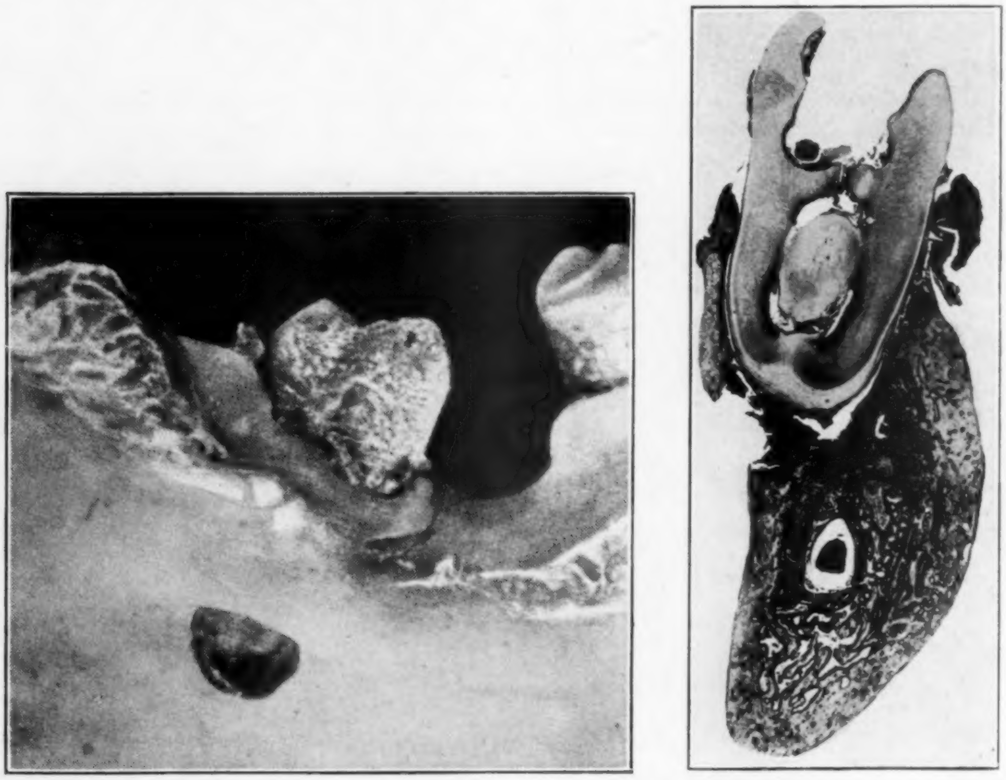
In these 24 cases the first molar tooth was involved six times, the second and the third molar tooth each twelve times. In other words, the percentage of the total of 30 molar teeth involved is for the first molar tooth 20, and for the second and third, 40 each.

These clinical figures present a striking correlation with the anatomic data given in foregoing paragraphs. Nevertheless, I do not wish to attach too much importance to these figures. They are intended to be taken not as proof but as supporting evidence.

Autopsy Observations Reflecting These Anatomic Statements.—Finally, I attempted to see whether these anatomic statements would be paralleled by corresponding observations given in reports of autopsies. However, there are comparatively few reports of autopsies in cases of Ludwig's angina, and most of the reports do not record the condition of the jaw with the exactitude that might be desired.

Ludwig,[16] for example, in reference to the autopsy in a case of his stated: ". . . The periosteum on the inner surface of the jaw was loosened from the bone and discolored. It was at this particular point that the gangrenous ichor first appeared." Unfortunately nothing is mentioned as to the exact location of this point.

Roser[7] made a reference to the report of an autopsy performed by von Recklinghausen. A "foyer purulent" was found starting from an abscess on the root of the fourth back tooth. (This very likely means the second molar tooth.) An autopsy reported by Thomas[11] revealed "on the internal surface of the inferior maxilla a fistula starting from the second molar tooth communicating with the gangrenous focus." Unfortunately, neither of them mentioned any relation of the fistula to the mylohyoid ridge.



A. *B.*

Fig. 4.—(Richard's[40] case). *A*, perforation of the inner plate of the mandible caused by caries of the second molar tooth. It is located below the mylohyoid ridge. The root of the molar tooth is visible in the hole. *B*, perforation of the inner wall of the mandible at the site of the second molar tooth.

The findings of Richard[40] in an autopsy performed by him seem to me to be much more interesting and particularly instructive.

". . . The exact dissection of the neck tracing the gangrenous channels of the tissues leads toward the region of the second lower molar. The periosteum of the mandible is discolored and loosened. After the removal of the periosteum there is found lingually immediately *below the linea mylohyoidea* at the compacta of the bone a hole of the size of a pepperseed. Within this hole the tips of the roots of the molar were distinctly visible. The tooth (second molar) shows a carious cavity."

Fig. 4 is a reproduction of two illustrations belonging to Richard's[40] article. Fig. 4, *A* is instructive because it shows the hole below the mylohyoid ridge and the root tips plainly visible within this hole. Fig. 4, *B* shows the defect at the inner alveolar wall communicating with the carious cavity.

Fig. 5 is a photograph of a diagram from the article of Trautmann.[15] It shows (in contrast to Richard's case) a hole in the compacta of the mandible above the mylohyoid muscle, starting from the root of the first premolar tooth. The result was, of course, sublingual cellulitis spreading toward the epiglottis and even to the contralateral side. The structures below the mylohyoid muscle were not affected at all.

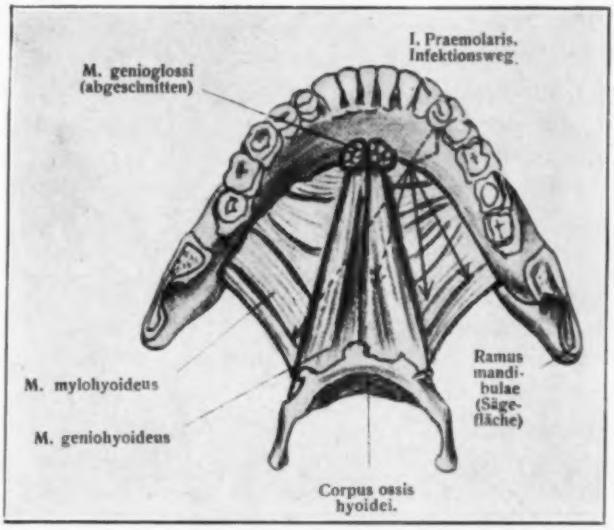


Fig. 5.—Diagram from Trautmann's[15] case. The infection started from the first premolar tooth. Spreading occurred above the mylohyoid muscle.

I call attention to these reports because they seem to me in a certain sense a remarkable pathologic and clinical reflection of the anatomic findings as described on a foregoing page. Moreover, they bring out the importance of exactly observing at autopsy, in cases of Ludwig's angina, the condition of the mandible and especially the state of the molar teeth and the site of a fistula, which apparently sometimes escapes observation unless the swollen and discolored periosteum is removed. A better understanding of the genesis of this disease will contribute to its prophylaxis, diagnosis and therapy.

SUMMARY

A brief review of the history of Ludwig's angina is given. According to Ludwig's description, this entity is characterized by gangrenous cellulitis originating in the submandibular (submaxillary) space and rapidly spreading toward the floor of the mouth.

Neither isolated cellulitis in one of these spaces nor suppurative submaxillary lymphadenitis can be regarded as Ludwig's angina. For the second condition which sometimes appears as a comparatively benign imitation of Ludwig's angina, the term "pseudo angina Ludovici" or "pseudo Ludwig's angina" is suggested.

The frequency of the dental origin and the almost exclusive involvement of the molar teeth in Ludwig's angina are emphasized on the basis of data compiled from the literature.

The incompetency of the explanations usually given for the predominant role of the molar teeth in the genesis of Ludwig's angina is shown particularly on account of anatomic studies.

In an attempt to find a solution of the problem of the genesis of this disease, anatomic examinations were undertaken to determine the relation of the roots of the molar teeth to the mylohyoid ridge. These showed that the second and third molar teeth almost invariably reach as for as, or even below, the mylohyoid ridge, that the first molar tooth in most instances reaches a point above the ridge, and that the tips of the roots of the anterior teeth are exclusively above this ridge. These findings explain the fact that an infection arising from an infected molar tooth, especially a second or a third one, usually extends into the space below the mylohyoid muscle.

Statistics concerning the involvement of the three molar teeth in the etiology of Ludwig's angina were compiled from the literature. They show a striking similarity to these anatomic findings.

Attention is called to the report of an autopsy in a case of Ludwig's angina, which showed a perforation of the inner plate of the mandible below the mylohyoid ridge which resulted from a carious process in a second lower molar tooth. In another case, in which the infection started from a premolar tooth, the perforation was located above the attachment of the mylohyoid muscle. The result here was sublingual cellulitis. These reports are recalled because they represent clinical support for the anatomic findings mentioned.

I particularly wish to thank Dr. Samuel Iglauer for his aid. It is a pleasant duty for me to acknowledge his interest in the problem, his many helpful suggestions and objective criticisms. I also wish to express my gratitude to Dr. Samuel Brown and Dr. Archi Fine for making the x-ray pictures.

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EARLY TREATMENT OF BURNS

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Burns, either alone or in combination with other injuries, are a frequent cause of casualties, both for civilians and members of the Armed Forces, during the present war. As most individuals thus injured live, mortality rates do not give a true picture of the economic loss. The price of prolonged hospitalization, suffering, disability, disfigurement, and mental upset is enormous. Members of the medical profession now in the Armed Services, who in ordinary circumstances would have referred serious cases to those particularly interested in this subject, now must undertake their management alone. This problem would not have been difficult if a burn were an entity like many other conditions. On the contrary, it requires knowledge of the treatment of a peculiar type of large wound. It is made doubly difficult by being a wound which is extremely painful, usually has copious exudate, is accompanied by much edema in adjacent tissues, is easily contaminated, and which cannot be closed immediately unless small. If treated kindly, it will close itself quickly by the natural healing processes, when the injury has been superficial. If deep, delayed closure takes place by healing from the sides of an ulcer with contracture and scar epithelium, or by skin grafting. The early treatment has many controversial aspects about which there is no general agreement.

The Problem.—It is not the purpose of this communication to discuss the relative merits of innumerable agents and methods used in local treatment. One method known to be good will be discussed and described. This decision was made because there is need to urge one form of local treatment which is so simple that it can be understood and put into reasonably good practice by those who, having never treated a severe burn previously, are now faced with the possibility at home, and the probability in battle areas, of having to care for many such casualties. The members of the profession who treated large numbers of burns in peacetime were few. Suddenly it has become necessary that many be prepared to do so. The multitude of materials, and the methods for their application, which have been advised (and to which there is constant addition), has served to confuse the uninitiated who are to be the all-important vehicles for carrying early treatment to the patient. This is particularly so in the Armed Services. Those who have special knowledge and facilities should undoubtedly continue with investigation of the burn problem in all its aspects. One part of this is the finding of better materials and methods for local therapy.

The subject of burn treatment may be divided into that designed for prevention and that designed for cure. That "prevention is better than cure," is most true when it is applied to burns. Much remains to be done on this aspect

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of treatment which is not within the scope of this communication. Therapy designed for cure is divided into early, intermediate, and late. The early treatment may be said to have ended when the first week has passed. This time is chosen because then the initial phases of primary and secondary shock have elapsed. The liver and kidneys are beginning to show evidences of recovery from the toxic state.[1] The average time of death in our cases in recent years has been about five days (excluding deaths later than ten days after the accident).[2] This article will discuss early treatment under the heading of local and general therapy. It is not intended that anything original be introduced. The material presented represents opinions formed as the result of experience with many burns in children and adults in civilian and service practice.

Topical Application.—The question of local application, being the most controversial, is discussed first. In choosing a material for service use, simplicity must rule. It is better if it is the same for first aid and definitive therapy. Whatever is chosen should be available in large supply, easily packaged for continued sterility and handiness in use, and capable of withstanding wide variations in temperature without change in composition or texture on return to ordinary operating conditions. Its use should be easy for nonmedical personnel and untrained medical personnel. It should not be harmful to already damaged tissue cells. The latter is one of the controversial points. Except in a gross manner, it is difficult to know what materials are injurious when applied directly to the exposed epithelial cell elements remaining in the dermis. The healing processes are undoubtedly affected by the local application, but controls are difficult to arrange, and the assessment of results to the satisfaction of everyone is practically impossible. The presence of infection, maceration, and chemical irritation or necrosis are the most obvious causes of damage to epithelial cells during the course of treatment. They are likely of greatest importance in the order named. The combination with the material used for local application of bactericidals or bacteriostatics would appear to be a sound principle as long as these substances do no direct damage to the remaining dermal epithelial cells, or the course of repair. The latter includes the separation and removal of dead tissue, and the recovering process. The details of how this takes place, and what retards or expedites it, are so little understood that it is useless to wait for decisions on controversial points to determine what should be urged presently for local application. Time will bring the answers to many questions, but that does not help those whose problems are urgent today. This pertains particularly to those in the Armed Services unskilled in the management of burns. They must be given simple directives to the query, "What is the best method of handling the burned area?" That which constitutes the "best" must at most represent an opinion with which many may not concur. Thus a "good" method as viewed by present-day standards, and one which uses a material for local application which satisfies reasonably well the desirable features mentioned above, is a cream, jelly, or ointment ***occlusive pressure dressing method*** such as described by Koch,[3] Ackman and Wilson,[4] and others.

Consideration of a Cream, Jelly, or Ointment.—The perfect material for local application has yet to be found. There are so many qualities that are expected of it that the likelihood of combining them in one material is remote. The

search has received most decided impetus since the commencement of the war. The comparison of materials is difficult owing to the varied depths of burns, unknown factors affecting healing, the myriad permutations and combinations of differences in age, sex, race, nutrition, hormonal activity, etc., which make one individual different from another. Many substances have had extravagant claims made on their behalf, such as vitamins and hormones in mixtures. There is lacking anything approaching conclusive evidence of their value. The "kindliness" with which some donor areas heal after removal of "calibrated" skin grafts, when compared to other parts of the same area treated differently, is fair proof of the undesirable qualities of some forms of surface application.

The advantages of an ointment, cream, or jelly are many. Those of particular importance are listed.

1. It is easy to apply by medical and nonmedical personnel.
2. It needs no elaborate paraphernalia in its employment.
3. It can be packaged in a sterile fashion in suitable containers.
4. It can be combined with gauze mesh in the original packaging. This eliminates one maneuver in the primary dressing.
5. Depending on the medicaments in it, it can be made available in large supply.
6. Ointments can be made, which after wide variations in temperature, return to the original composition and texture.[5]
7. It can be applied quickly—time does not have to be allowed for drying as in the case of escharotics and plastic materials.
8. A repeat application is not necessary. The first-aid treatment can, if circumstances demand, become the definitive treatment.
9. Nursing care necessary is at a minimum.

All of these points are most advantageous in service practice. Indeed, they are in certain situations "all important."

Ointments vary much and that which is an advantage in one is a disadvantage in another. A water miscible base would appear to have some advantages, if the material for local application were to be changed to another whose action was disturbed by the presence of oil. If the original material were continued to the skin grafting stage it would be necessary to switch from the oil containing ointment. However, this is a minor matter as the patient is by that time probably in a hospital where such changes are not difficult. Obviously, there is much to be learned concerning the bases from which ointments are made and their effects on the repair processes. That infection and maceration are harmful has already been stated. Skin surfaces may have epithelial cells injured by the steeping which takes place when a dressing is used which prevents the escape of moisture. This is prone to take place under vaseline, but less so if it is combined with other medicaments. Thus, borated vaseline seems less macerating. Water miscible creams and jellies are less likely to cause it.

Contamination and Infection.—The problem of infection is best managed by prevention of initial contamination rather than by bringing it under control later. This prevention may be impossible due to circumstances, such as the nature of the injury, and the time between the accident and satisfactory therapy. Infection means the invasion of the tissues of the body by living pathogenic

organisms in such a way as to favor their growth and permit their toxins to injure. Thus, there is often contamination without the condition progressing to an infection of any significance, despite the fact that this large open wound has some partially cooked tissue as its base, or later has granulation tissue. Infection in our experience has not been a great producer of morbidity or mortality. That contamination can be completely removed or infection can be controlled by surface applications of medicament is controversial. The type of medicament, the amount and type of contamination, and the quantity of necrotic tissue present would appear to be important factors. Sulfonamides have been of help. Other bacteriostatics and bactericidals of more ancient use are also of value. There has been some tendency to put them aside. That sulfonamides represent only a phase in the search for better materials is forecast by reports of penicillin as published.[6] While sulfonamides are far from ideal, their known value at present is sufficient to cause their inclusion in any ointment which is chosen, until other compounds have been well tried and found more satisfactory by those who have proper skill and facilities. The absorption from a large open wound such as a burn of second degree is so rapid that care must be taken not to employ a very soluble sulfonamide directly on the surface, or to incorporate it in an ointment from which it is not too quickly removed. Under such circumstances the toxic effect of the drug is an added insult to an already sick patient. Local concentration can be maintained for many days if sulfathiazole is incorporated as recommended by Ackman and Wilson.[7]

The Application.—When an ointment or cream is put on a burned surface it should spread easily and adhere to that surface. This is a quality not possessed by some unguents. This factor can be circumvented by applying them directly to the gauze, and thence to the body surface. It is even better if the material is parceled in such a fashion that this maneuver is not necessary, the ointment being impregnated in gauze and packed so that a small part may be used without gross contamination of the whole bundle.

The gauze may be of wide or close mesh. The former is preferable as it carries much ointment and allows drainage into supporting dressings. When granulation tissue forms the surface, the gauze should be finer as the granulations tend to grow through the coarse variety if it remains on for some time. The chief difference between this and *tulle gras* is in the material (ointment or cream or jelly) used with the gauze.

The local application should overlap considerably the limits of the burned area. It is padded by sterile fluffed gauze or machinist's waste. Firm bandaging with pressure reinforced by adhesive plaster fixes the dressing. The type of bandage used by us has been flannelette cut on the bias. This has some stretch so that pressure can be applied and maintained for some time. Elastic bandages or elastic adhesive rolls are satisfactory. Enough padding is used to absorb considerable exudate. More is necessary with superficial than with deep burns as the latter do not "weep" so much. The pressure is designed to check the "bleeding" of the plasma into the tissues adjacent to the surface lesion. Thus the local edema is largely controlled. If this pressure dressing is removed at any time during the first few days after the accident, swelling will take place.

The padding, and bandaging, and fixing of the dressing represent the most difficult part of this form of treatment. The supply factor (of padding and suitable bandages) is a disadvantage in service practice, but is not a great problem in civil establishments. It requires practice for medical personnel to put a satisfactory dressing of this type on hands or face where many of the burns occur. Nonmedical personnel would need much instruction and practice. Cleansing and débridement, local application, and the pressure dressing being three distinct steps in the therapy of the local lesion, the first and last may have to be neglected, from force of circumstance. The pressure bandage should be applied as soon as possible, and thus nonmedical personnel should be taught its use. Cleansing and débridement are closely similar to a surgical operation in that they need sterile supplies and sterile technique as well as a plan of action to produce the appropriate effect. Thus, first-aid therapy includes an ointment or cream in gauze application, next to the burned surface, overlaid by some padding, the whole being fixed by a pressure bandage and adhesive plaster. The first-aid therapy should be continued as the definitive treatment if much time has elapsed between the two, no débridement being considered advisable.

Too much emphasis cannot be placed on the prevention of contamination. Treating the burn as a large open wound if circumstances permit is most important. All the usual surgical precautions concerning sterility are urged—sterile gowns, gloves, drapes, instruments, and the masking of the patient and all who are near. If circumstances permit segregation of patients, burns should be in a ward apart from patients with infections. The local application, if and when it represents the definitive local treatment, should not be removed for at least a week unless circumstances warrant a change. The constant change of dressings does no good, and often does harm.

Cleansing and Débridement.—Controversy arises from the fact that the value of a débridement in this type of wound is not agreed upon by all. Certainly in many cases treated by us without débridement the results have been generally satisfactory. However, it is our opinion that débridement should not be abandoned by the casual treater of burns, until more evidence accumulates to support this contention. If there is no supply of sterile water, soap, or detergents, no method of controlling pain during the procedure, or much time has elapsed between the accident and the first treatment, the circumstances might justify its omission. This step in the treatment has been accomplished in our work by flooding the part with sterile water or saline solution. Cultures of the soap solution distributed by the pharmacy at our hospital resulted in the growth of a variety of organisms. Thus, a sterile solution of a soft soap is used. Benzine may be used at this stage to remove oil or grease. The débridement consists in removal of any portion of the skin that is loose or raised in blebs. This is done rapidly, using wet gauze as a "wipe." In the average case (less than 10 per cent of the body surface affected) it should not take more than two or three minutes. It may be necessary to scrub with a brush a burned area into which foreign particles have been driven. There must be some definite reason present for such therapy because it is associated with considerable direct trauma to the surface. The area is dabbed dry with a towel and the local application applied.

GENERAL THERAPY

Control of Pain.—While control of pain is primary in the mind of the patient, it cannot be carried out satisfactorily if there are no medical personnel present. Thus, the question of local application was dealt with first. Pain is controlled by intravenous morphine; $\frac{1}{6}$ gr. intravenously and a similar amount hypodermically by withdrawal of the needle is an average initial dose for adults. The intravenous portion is administered slowly. If medical personnel are not present and morphine is available it may be given ($\frac{1}{4}$ gr.) by mouth, but the delay in action is so great that if medical personnel can be reached within one-half hour it is better to give nothing. A general anesthetic is rarely necessary. Many burns are associated with damage to the respiratory tract by fumes or superheated air. Chest complications may occur rapidly. A general anesthetic is a complicating factor and should be particularly avoided in this type of case.

General Supportive Measures.—While primary or neurogenic shock may be seen, it has not been a factor in our cases. Lying down, with freedom from disturbance and maintenance of body warmth, should bring it under control quickly.

Happily, the subject of this article does not permit a discussion of what is meant by secondary shock or its signs and symptoms. Its treatment in burns of moderate or severe degree should start long before it can be seen clinically by ordinary observation. That is the first factor to recognize. The second is that it should be adequate. Whole blood, concentrated serum, normal serum, and plasma have been used by us in this phase. The viscosity of the concentrated serum made it difficult to employ. While some reactions (chills and fever), related by us to the administration of these materials, have been noted with normal pooled serum and pooled plasma, they have both been satisfactory within the limits of this type of therapy. Whole blood was used much, a number of years ago, and was also of undoubted value. As the red blood cells do not appear to be necessary or altogether desirable at this stage, plasma or serum are recommended. The amount of plasma necessary to control the hemoconcentration as evidenced by whatever estimation that is being used (hemoglobin, specific gravity, etc.) is large and bears some relation to the size of the burn. Ideally, to restore the original blood volume is the aim. From a practical point of view this should remain the aim if circumstances and supplies exist. Clinical improvement in patients with much smaller doses is remarkable, even without laboratory evidence of control of hemoconcentration.

Experience has taught that small burns (under 5 per cent) are not to be feared as far as mortality due to secondary shock is concerned. These, despite a degree of hemoconcentration, may be neglected as far as the attempt to maintain blood volume is concerned. Burns of over 10 per cent of the surface may be considered progressively more serious according to size. The necessity to provide agents to maintain blood volume also becomes increasingly urgent. As the flow of plasma into the tissue, and possibly from the surface, starts immediately, the intravenous plasma or serum should be started at once, without waiting for signs of distress on the part of the patient as evidenced by ordinary observation. Thus, the quantity of plasma is given empirically, additions being made as tests reveal the necessity. The majority of our patients having been

children, we have worked on the basis of 3 c.c. of plasma per 1 per cent rise in hemoglobin per ten pounds of body weight. If later the serum protein drop is significant or the hemoconcentration is not controlled, more plasma may be given. It is not our opinion that the apparently ideal quantity (i.e., the quantity necessary to completely restore the original blood volume as estimated by blood examinations, hemoglobin, hematocrit, etc.) must necessarily be given. Indeed, the use of such quantities might be criticized as wasteful of material which is expensive, and which on administration is not entirely free from reactions of a disturbing character. Whether plasma serum or whole blood should be given, and in what doses, for intermediate or late therapy, need not be discussed in this communication.

It is only fair to state that while quantities given at the present time might on occasions seem unnecessarily large, the error occurs more often in the opposite direction. Many rules have been given by which the quantity may be estimated, such as by Harkins,[8] Black,[9] and Elkington, Wolff, and Lee.[10] This type of supportive treatment is commenced immediately in a burn of over 10 per cent in an adult. In smaller burns our policy has been to be guided by the clinical data as they developed. The intravenous apparatus in our cases has been kept running between the periods when the serum or plasma was being administered, with 5 per cent glucose or a mixture of glucose and normal saline solution. It is most important that fluid output (urine and vomitus) be carefully measured. In an adult a twenty-four-hour urinary output of at least 1,000 c.c. is desired. For some hours after the accident, even in severe burns, the patient may take fluids well by mouth.

Concerning other supportive general therapy, the use of cortin has seemed in our experience to be of value. There is little beyond impression to support this, however. Its employment can safely be left with those conducting investigation. Its expense and scarcity are too great for routine use. Oxygen in high concentrations should be given to patients showing cyanosis. A slowed peripheral circulation and cyanosis is present in most cases of moderate or severe degree. There has been a tendency on our part to neglect this aspect of treatment.

Early Complications.—Mild jaundice is noticed occasionally. Beyond the usual supportive measures with glucose solution intravenously, no specific measures aimed at the cause of the lesion have been used by us. Vomitus containing changed blood is common. Massive hemorrhage may happen from erosion of the lower part of the esophagus or from stomach or duodenal ulcer; this is uncommon. Whole blood replacement is given. Uremia and anuria may occur and beyond the measures usually taken for these conditions nothing further has been employed. Chest complications due to associated respiratory tract damage must be dealt with as they arise. Oxygen is valuable. Two cases of mediastinal emphysema have been seen. In one no treatment was instituted and recovery took place. In the other case there was steady progression of the emphysema up over the neck and chest. A tracheotomy was performed with temporary improvement. The patient later died, and the value of the tracheotomy was problematical. Pneumonia has been seen in a number of cases. Acute

pulmonary edema has also been encountered. The giving of the large quantities of intravenous fluids (plasma and electrolytes) in such cases has been discontinued.

CONCLUSIONS

1. An attempt has been made to indicate one good method of early treatment so that the uninitiated faced with the likelihood of having to treat cases may be prepared both with the knowledge and the equipment to use one method well.

2. Emphasis has been placed on using the same materials and equipment for both first-aid and definitive treatment.

3. The topical application was discussed and the reasons given for the choice, with emphasis on simplicity.

4. The general measures associated with relief of pain and supportive measures were mentioned briefly. These have been discussed so much in recent literature that it appeared useless to go into detail.

5. The early complications were mentioned briefly.

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Editorial

The General Care of the Patient

PHYSICIANS consider supportive treatment to be of the greatest importance in cases of infection and serious wounds. Dentists, as a group, are weak to the point of being negligent in the general care of patients presenting lesions in the mouth, or infections of the jaws. It is true that the dentist is chiefly occupied with the filling and restoring of teeth. However, the large number of patients seen in hospital clinics with cellulitis, submaxillary and cervical abscesses is proof that such cases are, in general, poorly handled by the general practitioner of dentistry. In some cases, the local treatment is inadequate or too long delayed. Failure to remove the cause promptly, or to establish drainage in cases of an alveolar abscess, is frequently a reason for complications; many cases, however, go wrong because of failure to give supportive treatment.

What may we accomplish by supportive treatment? We simply intend to restore and maintain optimal physiologic conditions in order to promote the process of healing.

How do we determine the supportive substances that may be needed in a specific case? We make a clinical appraisal and use all available laboratory tests.

In a recent paper, Hoxworth* discusses methods used at the Cincinnati General Hospital, in an approach to the problems which arise in the supportive management of patients. The following statements are, in substance, taken from this timely article.

WATER BALANCE

Dehydration very commonly accompanies shock, but loss of water, proteins, and electrolytes occur in all severe injuries, as well as after prolonged periods of anesthesia, vomiting, and diaphoresis. Water requirements must be met to prevent tissue dehydration and to secure a correct water balance between the tissues and the circulatory system. Besides physiologic plasma, protein and chloride levels must be maintained. Optimum water balance is a prophylactic measure against delayed wound healing and wound infection.

The degree of moisture of the skin, tongue, and lips gives an indication of the state of hydration, and the measured and recorded water intake and urinary output furnish an index of importance. A urinary output of from 1,000 to 1,500 c.c. in 24 hours indicates adequate hydration in the adult whose kidney function is not damaged. If the patient receives sulfonamides, the establishment of a good urinary output is important in avoiding urinary complications.

Depleted protein reserves are indicated by low plasma protein percentages (normal 6 to 8 Gm. per 100 c.c.). Plasma is given if the hematocrit deter-

*J. A. M. A. 124: 483, 1944.

mination is normal (42-50 per cent in the male, and 39-43 per cent in the female, by the capillary tube method of Guest and Siber) or elevated, but if it is subnormal with low or normal plasma proteins, whole blood is administered.

Solutions of electrolytes in the form of 5 per cent dextrose in saline solution, or dextrose in distilled water, are given intravenously in sufficient quantity to maintain adequate urinary output and normal plasma chloride levels.

NUTRITION

The nutritional requirements are of great importance. An adequate supply of dextrose, proteins, and vitamins aid in general recovery, promote healing, and combat infection. Holmes* in an article on wound healing recommends as an optimum diet for patients with infections, one of high calorie, high polyvitamin content, and the protein fraction raised at the expense of fat. Patients who cannot take food by mouth should receive dextrose intravenously in a 5 to 10 per cent solution, not faster than 500 c.c. per hour. Protein is supplied by the administration of 500 c.c. of plasma per day (human plasma contains about 70 Gm. of protein per liter).

Vitamins are of greatest value in wound healing, and in promoting resistance to infection. It should also be remembered that vitamin reserves are quickly depleted in surgical infections. If patients are fed parenterally large amounts of dextrose for caloric requirements, they should receive thiamine, riboflavin, and niacin in the ratio of 1:2:10, according to the Council on Foods and Nutrition of the American Medical Association.† For maintenance, 2.5 mg. of thiamine, 5 mg. of riboflavin, and 25 mg. of niacin is considered sufficient. Vitamin C is important because of its contribution to the formation of intercellular substance (collagen and osseomucin); 100 to 200 mg. per day should be given. Vitamins A and D are given as indicated in case of manifest deficiencies. Vitamin A deficiency may predispose to intercurrent infections.

ANEMIA

Anemia may be due to loss of blood, or may be secondary to sepsis, or may have existed previously. Since poor oxygenation of tissues will delay wound healing, anemia should be treated by whole blood. One pint of whole blood can usually be depended upon to elevate the red blood cell count by about 400,000 cells, and the hemoglobin by 1.1 Gm.

REST AND IMMOBILIZATION

Bed rest is one of the most important factors contributing to prompt recovery of the patient, and is the one most commonly disregarded by the dentist and even by many of the oral surgeons. Just is important is immobilization. Rogers‡ states that the broad principle involved in immobilization is the curative effect of rest; it controls pain, absorption, and subsequent trauma. Immobilization allows the local muscle and sympathetic vascular spasm to subside; it helps to decrease edema and the lymph flow; and thus the absorption of toxic

*New England J. Med. 227: 909, 1942.
†J. A. M. A. 119: 948, 1942.
‡New England J. Med. 229: 218, 1943.

substances resulting from tissue damage, of bacterial toxins, and bacteria themselves, is reduced. This gives the blood stream a chance to neutralize and eliminate them, and thus minimize toxemia.

CHEMOTHERAPY

Everyone is well acquainted today with the use of the sulfonamides by oral or intravenous administration, as a protection against infection when operating in the presence of sepsis, or in inhibiting bacterial activity in wounds or abscesses. Anyone prescribing these drugs, however, should keep in mind the dangers and contraindications of their use. Patients receiving chemotherapy should be hospitalized, and under constant observation. Blood cell counts and blood level determinations of the drug should be made frequently; kidney function must be watched. It is generally felt that the main benefit of chemotherapy is to prevent the spread of infection.

Penicillin, though not as yet in general use, has been obtainable for investigative purposes. The importance of supportive treatment when penicillin is used is stressed by Lyons,* especially in cases of nutritional depletion. The nitrogen balance should be maintained by supplying protein, either by plasma which contains 7 Gm. protein per 100 c.c., or whole blood which supplies more nearly 18 Gm. per 100 c.c. The requirement is generally met by 2 liters of plasma, 750 c.c. of whole blood, or a mixture of 500 c.c. of whole blood and 500 c.c. of plasma. Because of the greater need for hemoglobin, the use of whole blood is preferred. Such therapy is useful in preparation of the patient for operation, as well as during convalescence to maintain blood volume, hemoglobin, and red cell values. Transfusions of whole blood, in large quantities, are particularly indicated on patients in whom active infection is associated with nutritional depletion and anemia. Lyons estimated that 1,500 to 3,000 c.c. of blood per patient is required.

—*K. H. T.*

*J. A. M. A. 123: 1007, 1943.

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THE CLINIC OF THE DENTAL DEPARTMENT OF THE MASSACHUSETTS GENERAL HOSPITAL AND THE DEPARTMENT OF ORAL SURGERY, HARVARD SCHOOL OF DENTAL MEDICINE

Volume II

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INTRODUCTION

WE ARE presenting herewith the second Massachusetts General Hospital Number. It contains cases treated in the second half of 1943. A great many face and jaw fractures have been included, because these injuries are of particular interest in wartime. Many of our cases are being treated by the well-known, conservative methods. For this publication, we have selected only the unusual injuries, and those in which the use of skeletal fixation and internal wiring were thought to be indicated. Of 67 patients treated for fractures in 1943, skeletal fixation was used in 19 per cent, and internal wiring fixation was resorted to in 6 per cent.

Osteomyelitis is quite frequently associated with fractures, especially those compounded into the mouth and involving a tooth or several teeth. Skeletal fixation has been used in several such cases with success. In osteomyelitis with fracture, or fracture with osteomyelitis, special attention must be given to the proper placement of the half pins to prevent spreading the infection. A case presenting various additional complications will be presented.

The removal of foreign bodies also plays an important part in civilian as well as in military injuries. Aids for their quick removal should be helpful. A method employed to remove a needle is included.

Permanent anesthesia and paresthesia often result from facial injury, especially in mandibular fractures with malunion. A method is described which promises relief for many patients so afflicted. Neurorrhaphy has been developed by the neurosurgeons to a fine art, and there is no reason why their methods should not be applied to the mandibular nerve.

A case of Paget's disease involving the upper jaw is recorded so that it may be compared with a case of fibro-osteoma which resembled the former in some respects, both clinically and roentgenologically. Many cysts and tumors are constantly seen in the clinic. Large cysts, when expanding, produce

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tumorlike swellings that may be mistaken for neoplasms. One of the cases included also presented an atypical x-ray picture. In another in which the x-ray looked like a cystic odontoma, we found that a definite adamantoblastoma had developed. Finally, a case demonstrating an interesting jaw deformity as a result of ankylosis concludes this series of reports.

I. CASES OF FRACTURES OF THE FACE AND JAWS

Case 24

Septic Compound Mandibular Fracture Complicated With Submaxillary and Pterygomandibular Abscess Treated With Penicillin

L. F. (No. 430632), a 70-year-old woman, was first seen on November 20 because of a partly erupted third molar in the lower jaw on the right side. For four months this patient had experienced pain and discomfort when eating.

Seven years ago she had her upper teeth removed and a denture made, which she wore with comfort. One year ago her remaining lower teeth were removed, and a denture was fitted which gave her good service until four months ago when she noted some pain on the alveolar ridge in the back of the lower jaw on the right. Here the gingiva had been perforated and the crown of an unerupted tooth was discovered by her local dentist. He found some pericoronal infection, which at the time was aggravated by wearing the denture.

Examination showed the patient to be in good general health for her age, but she said she was tired out and somewhat debilitated because she was unable to eat properly. For the past two years she had had arthritis of her hands, feet, and back. Her feet and back had improved, but her hands had become swollen and clumsy. She had some arteriosclerosis. The blood pressure was 165/90.

Local examination showed a perforation of the gingiva, through which the enamel of an unerupted tooth could be seen. There was some evidence of infection of the gingival margin, but no marked swelling or pus discharge. A smear showed many gram-positive cocci, and some pus cells, but no Vincent's organisms. An x-ray showed an unerupted third molar in a horizontal position surrounded by sclerotic bone; some osteolysis was present around the crown of the tooth, indicating infection. The patient was advised to have the tooth removed.

The patient was admitted to the hospital on December 3, and on December 4 the operation was performed with local anesthesia. Four cubic centimeters of monocaine, 1 per cent, with epinephrine, was injected for a mandibular block and local infiltration. An incision was made in the gingiva over the tooth, a gingival flap was dissected from the bone and retracted, and the bone overlying the tooth and that on the external surface was removed with bur technique and chisel. The tooth was ready to be removed, but it was impossible to dislodge it either by using forceps or the elevator technique. When greater force was applied, the tooth still refused to yield; instead, the jaw fractured beneath it. General anesthesia was then induced in order to reduce the fracture. An x-ray, taken with the endotracheal tube in place, through which the gas, oxygen and ether were administered, showed a horizontal fracture extending through the

ramus to the attachment of the stylomandibular ligament (Fig. 92). The tooth was cut away from the bone with rongeur forceps and preserved for pathologic examination. The incision was extended over the anterior border of the ramus, and a hole drilled from the outer to the inner surface, through which a stainless steel wire, 22 gauge, was threaded. This was finally passed around the inferior border of the mandible by means of the technique for introducing circumferential wires with a hypodermic needle. The wire was then drawn taut and twisted, the ends being pressed into the socket from which the tooth had been removed (Fig. 93). Sulfonamide powder was placed in the bone wound and the mucosa sutured with silk, after a rubber dam drain was inserted.

Pathologic examination of the tooth cut away from the jaw showed that it had been firmly ankylosed to the adjacent bone. Fig. 94 shows the distal surface of the mesial root with the bone (*B*) attached to the thick layer of cementum (*C*). Fig. 95 shows the mesial surface of the same root. The cementum has been resorbed and bone (*B*) has attached itself to the areas of resorption extending into the dentine (*D*). In both illustrations, the periodontal membrane which generally is found between the tooth and the bone—and ruptures when the tooth is extracted—has disappeared, and the cleavage occurred in the underlying jaw, as seen in Fig. 95.

Chemotherapy and Local Treatment.—The postoperative care included chemotherapy; sulfadiazine was given, 2 Gm. at the beginning and 1 Gm. every four hours. An ice bag was placed on the jaw for one-half hour every hour, and 1 grain of codein sulfate, with 10 grains of aspirin, was ordered p.r.n.

Some swelling developed in the submaxillary region the day after the operation. This was accompanied by a rise in temperature to 100° F. with a pulse rate of 100. The dentures, united by wires to form a splint, were to be inserted, but, because of intraoral swelling, this accessory immobilization of the jaw was temporarily abandoned. The following day, poultices were applied at the angle of the jaw, and on the third postoperative day there was some discharge of pus from the puncture wound made to introduce the circumferential wire. The sulfadiazine level was 6.4 mg. per cent. The third day, a larger incision was made. Irrigation with chlorazene showed that a communication existed between the abscess and the wound in the mouth. On the fourth day the patient felt more comfortable because of adequate drainage. Irrigations with chlorazene were continued, and an x-ray was ordered.

On December 9 the x-ray taken on the previous day was reviewed, and it was thought to show that the jaw was "maintained in position by the wire loop; there was no evidence of bone resorption or of osteomyelitis." On December 10 the pus discharge became more abundant, and on December 12 it was copious. A white count made on December 10 showed 18,400 white blood cells. The sulfadiazine level was 8.2 mg. per cent; the dose was changed to 1 Gm. every six hours. The temperature remained between 99.5° and 100.5° F., but the patient's general condition declined and she lost considerable weight despite accessory feedings of a high caloric diet rich in proteins and vitamins. On December 15, the hemoglobin was 12 Gm., and the white count 9,500 with 90 per cent polymorphonuclear leucocytes, 9 per cent monocytes, and 1 per cent

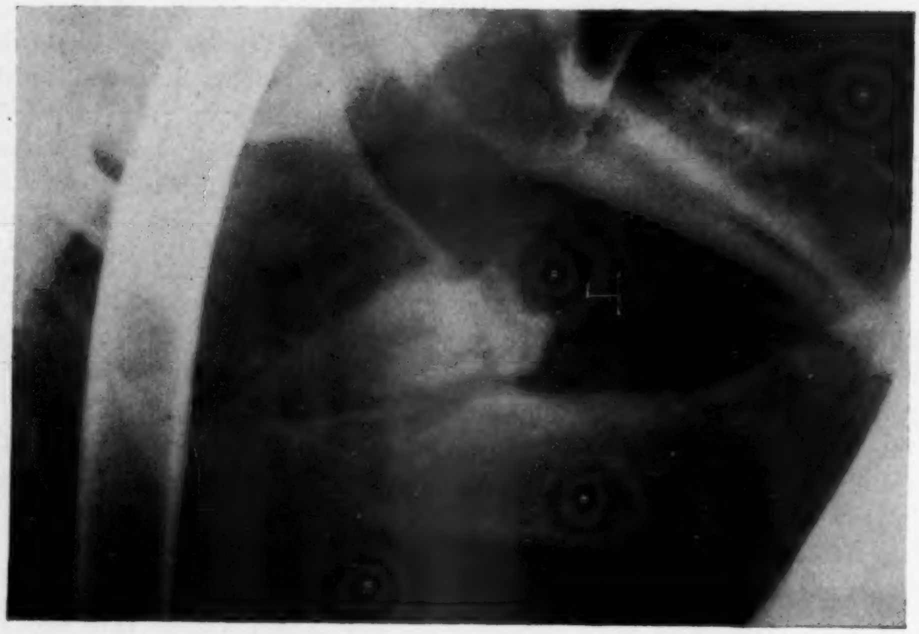

Fig. 92.—X-ray taken under general anesthesia. Note endotracheal tube, and fracture extending under the mesial surface of the molar to the posterior surface of the ramus. The tooth was ankylosed to the jaw.

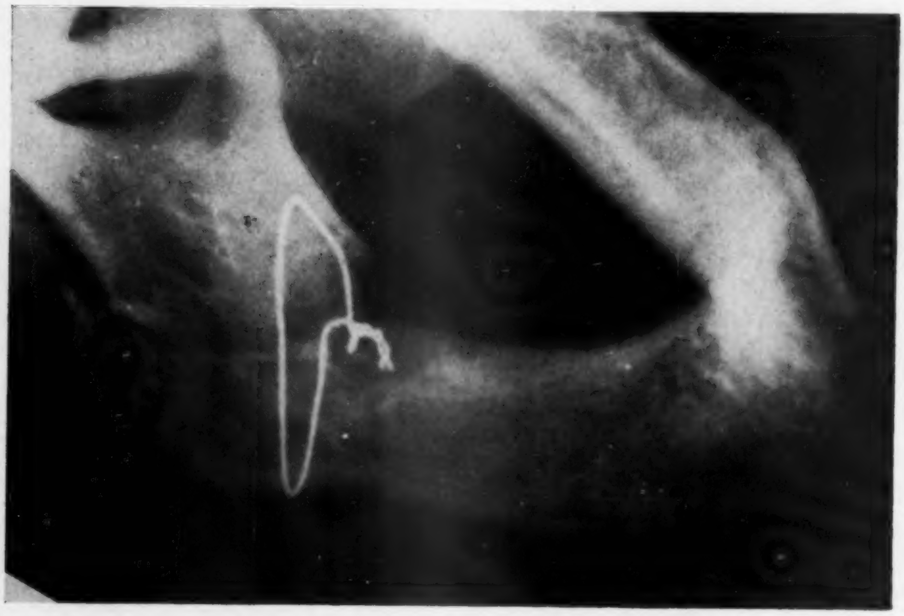

Fig. 93.—Postoperative x-rays showing reduction and internal wiring fixation.

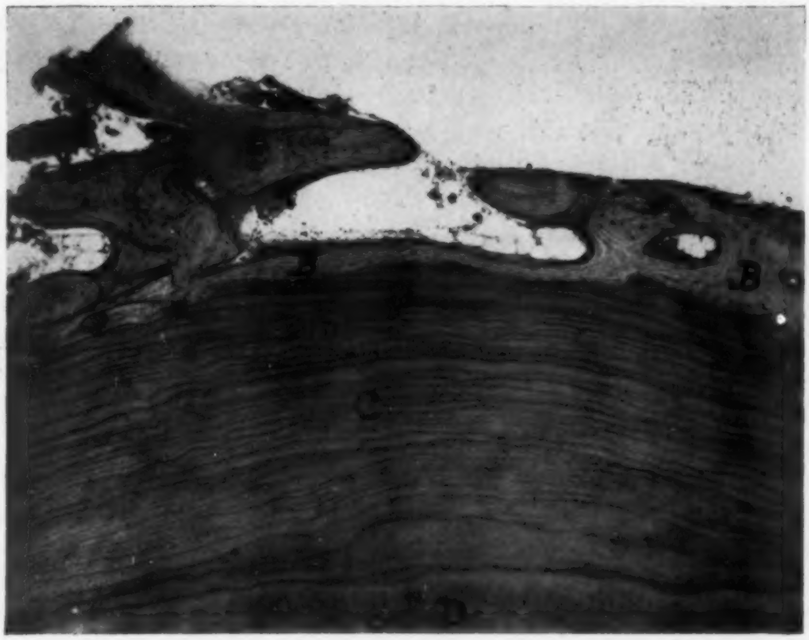

Fig. 94.—Photomicrograph of distal surface of mesial root showing ankylosis of bone, *B*, and cementum, *C*, of tooth. *D*, dentine.

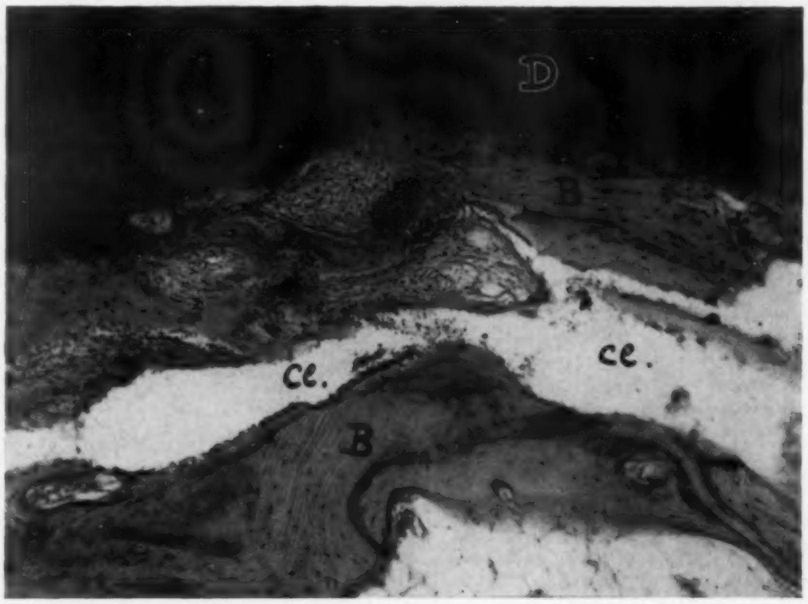

Fig. 95.—Photomicrograph of mesial surface of mesial root, showing ankylosis of bone, *B*, to dentine, *D*, which had undergone resorption after the cementum had been completely removed. *Cl* shows cleavage in underlying bone instead of between tooth and bone.

eosinophiles; red blood cells and platelets were fairly normal. The patient was given 500 c.c. of whole blood, which made a great change in the asthenia that had gradually developed. She was then given 200 mg. ascorbic acid daily, M.G.H. vitamin B complex 5 c.c., three times a day, and ABD capsules, one three times a day. Five cubic centimeters of the M.G.H. vitamin B complex consists of thiamine chloride, 10 mg.; B_1 complex, 3 mg.; pyridoxin, 1 mg.; pentothanic acid, 1 mg.; nicotinic acid, 20 mg.; and para-aminobenzoic acid, 1½ mg.

The profuse pus discharge continued, however. The temperature spiked between 100° F. and 102.5° F. On December 18, a Dakin tube was inserted through the incision into the submaxillary space, and by means of an intravenous drip, continuous saline irrigation was instituted. This brought about very little change in the local condition and was discontinued on December 22. At this time the sulfadiazine level was 9.5 mg. per cent, and the white count 16,800. On December 23 and 24 the patient complained of pain when swallowing. It was necessary to give her codeine and aspirin frequently, and hot compresses were applied to the jaw. On December 26 the lateral pharyngeal wall was seen to bulge out so as to occlude half of the pharynx, and it was clear that a pterygomandibular abscess had formed. The temperature had ranged between 101° F. and 103° F. since December 24, and it rose to 104° F. on December 28. The discharge from the skin incision was profuse, as well as that from the intraoral wound where the tooth had been removed, in spite of frequent irrigations with Dakin's solution through the inserted tube. Evidently the drainage was inadequate. The patient's blood pressure was down to 110/70.

On Dec. 28, 1943, with nitrous oxide, oxygen, and ether anesthesia, intratracheal method with pantopon and atropine for premedication, the incision at the angle of the jaw was enlarged so as to open wide the submaxillary space. The inferior border of the mandible was reached, and by finger dissection the bone was followed on the inner surface along the anterior border of the internal pterygoid muscle to the pterygomandibular space which was opened. Considerable pus was evacuated. The outer surface of the ramus was then explored, and after making a second incision further down on the neck at the place to which the infection had extended, a Penrose drain was inserted from the upper incision on the neck into the pterygomandibular space by means of a pair of curved hemostatic forceps. It was placed high enough so that the tip of the forceps could be felt when palpating the wall of the pharynx. At this time the mobility of the fracture was tested, and it was found that there was no sign of union. A second Penrose drain was then inserted through the lower neck incision to the outer surface of the mandible, after which a dressing and four-tailed bandage was applied.

The pus discharge continued to be profuse. The bacteriologic report from the culture taken during the operation showed no growth on the blood agar plate, but on plain broth *Staphylococcus aureus* was obtained.

In spite of the fact that good drainage now had been secured from both the inner and outer aspect of the fractured ramus, and repeated irrigations with Dakin's solution through a cannula inserted along the tubes were carried out daily, no headway was made in the treatment of the infection. Another

x-ray taken at this time "showed the previously described fracture which is fixed in position by wire. The position and alignment appear good. There is, however, a 2 cm. bone defect at the fracture site, but there does not appear to be any definite evidence of resorption about the wire. No sequestra are seen and there is no definite evidence of osteomyelitis" (Fig. 96).

On December 30 the sulfadiazine was discontinued since it seemed to have no beneficial effect, in order to determine more exactly the bacteriology of the infection.

Fig. 96.—X-ray showing an osteolytic defect in the fracture site.

On Jan. 2, 1944, a culture was taken from the pus discharging from each fistula, and given to Dr. A. B. Mangiaracine, the bacteriologist at the Massachusetts Eye and Ear Infirmary, who was to carry out the bacteriologic study with the consent of Dr. LeRoy A. Schall, the director. The following report was received:

Smear: Loaded with gram-positive cocci.

Culture: 1. Coagulose positive hemolytic, *Staphylococcus aureus.*
2. Alpha hemolytic streptococcus, micro-aerophilic.
3. *Clostridium spoorgenes.*
4. Gram-negative bacillus bacterioides.

The staphylococcus and streptococcus were penicillin 4 plus sensitive. A Petri dish inoculated with *Staphylococcus aureus* on blood agar, in the center of which was placed a small piece of filter paper moistened in penicillin (diluted 5 units in 1 c.c.), showed, after 24 hours' incubation, a halo around the filter paper caused by the bacteriostatic action of the drug (Fig. 97). The dish was left on the desk in the laboratory in a slanted position for another

Fig. 97.

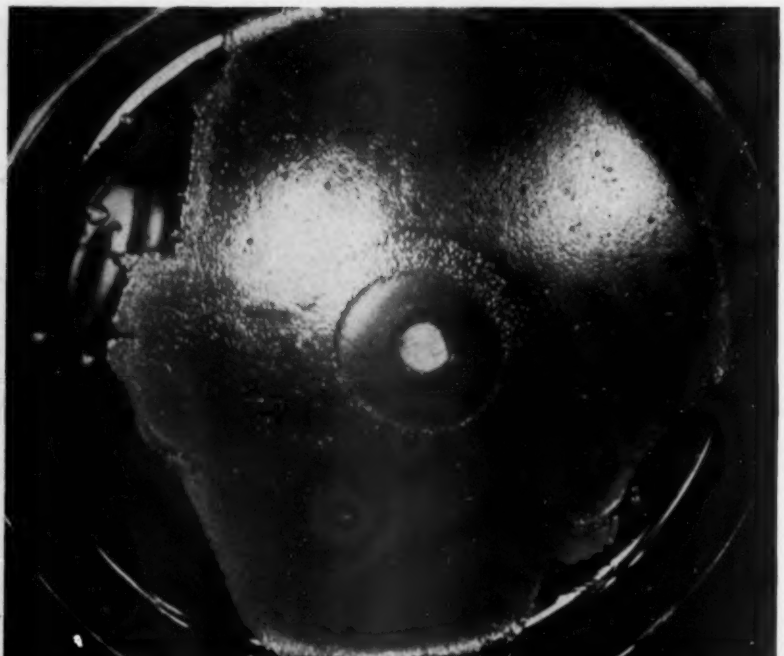

Fig. 98.

Fig. 97.—Blood agar plate with staphylococcus culture. Note inhibition of bacterial growth around filter paper saturated with penicillin.

Fig. 98.—Blood agar plate after forty-eight hours. When tilted, some of the penicillin gravitated from the original area and destroyed the culture in the second ring.

Fig. 99.

Fig. 100.

Fig. 99.—Dressing on the morning of January 3, when penicillin treatment was started. Note discharge from around Penrose drains and pus on dressing.

Fig. 100.—Dressing on the morning of January 5 still showed copious pus discharge and new fistula developing.

twenty-four hours, when it was found that through gravitation the drug had caused an extension of the area devoid of bacterial growth, indicating the bacteriocidal effect of penicillin (Fig. 98).

Fig. 101.—Dressing on the morning of January 11, after eight days of treatment with penicillin, was practically without stain. Bands around Penrose tubes clean.

Fig. 102.—Dakin tubes used for local treatment with penicillin inserted through incisions, one into pterygomandibular space, the other to outer surface of ramus.

Application was made for penicillin, and it was received from Dr. Donald Anderson, the associate of Dr. Chester Keefer, representative of the National Research Council in charge of the distribution of the drug. Dr. Anderson advised intramuscular injection, 2 c.c. every three hours, of a solution containing 5,000 units per 1 c.c. He dispensed 800,000 units, which was to last about ten days.

Penicillin Therapy.—The treatment was started on Jan. 3, 1944. The dressing on that morning had shown evidence of copious pus discharge, as indicated in Fig. 99. The next day there was not much change. The temperature continued to spike to 101.5° F., but from January 5 on, it ranged between 99° and 100° F. A new fistula had developed (Fig. 100); this was incised and drained and later connected with the main incision. A culture yielded a long chain streptococcus which was identified as an anaerobic nonhemolytic streptococcus. On the same day cultures from the other incisions yielded *Staphylococcus aureus* 4 plus, alpha hemolytic streptococcus 2 plus, and bacterioides 1 plus.

Fig. 103.—Barton bandage reinforced by elastics to hold interoral splints in place.

On January 7, the *Staphylococcus aureus* was reported 2 plus. The patient received a second transfusion of 500 c.c. of whole blood on January 8. On January 10, 11, and 12, only a moderate amount of *Staphylococcus aureus* was found; it was reported as 1 plus. On January 10 the white count was 11,600, and on January 11 a third blood transfusion was ordered, to give the patient's improved general condition another boost.

On January 10 the patient's physical condition and mental attitude were excellent. The dressing removed in the morning showed barely a stain, and the wounds around the Penrose tubes were clean (Fig. 101).

On January 12 the bacteriologic report from a drop of pus pressed from the incisions, which still looked well, was worse: *Staphylococcus aureus* 4 plus. On January 13 and 14, other organisms had returned; the report showed the

presence of *Staphylococcus aureus* 4 plus, alpha hemolytic streptococcus 2 plus, *Clostridium welchii* 4 plus, and bacterioides 1 plus. The white count was 7,700. A new x-ray study showed "that there has probably been some decalcification at the fracture site, but we are not justified in calling it osteomyelitis."

The return of the organisms without changing the clinical picture, which was excellent, could be ascribed to the resorptive process caused by infection in the fracture line, or to bacteria harbored in the gauze contained in the Penrose tubes. An additional 800,000 units of penicillin were received to continue the intramuscular injections, and a small amount for local use, in dilution of 500 units per 1 c.c.

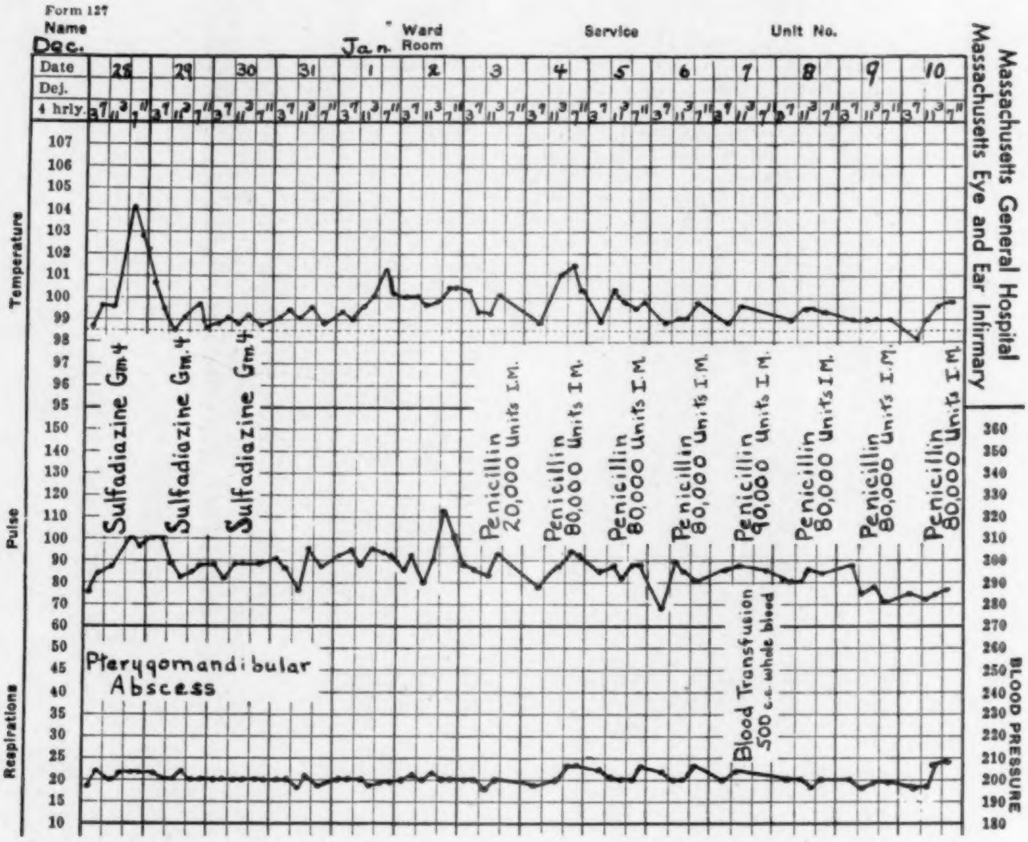

Fig. 104.—Temperature chart showing the last days of chemotherapy, and the beginning of treatment with penicillin.

The Penrose tubes were removed on January 18 under gas and oxygen anesthesia. There was hardly any pus discharge, and Dakin tubes were inserted and attached to the skin by means of silk sutures. The wounds around the tubes were packed with sterile gauze so that the penicillin solution injected would be more or less retained around the fracture site (Fig. 102). Three to 4 c.c. of the penicillin solution was injected into the Dakin tubes every three to four hours. A Gunning splint was improvised by wiring the patient's two dentures together. It could now be placed into the patient's mouth since all intraoral swelling had disappeared and the fistula at the extraction site had

healed. The splint was held in place by means of a Barton bandage reinforced by elastics (Fig. 103).

Cultures were taken every two to three days from the very scant seepage which could be pressed out from the incisions in the morning. All bacteria were again eliminated except *Staphylococcus aureus,* which persisted. A test showed that it was still sensitive to penicillin. On January 24 the white count was 7,900. The temperature curve remained flat (Fig. 104). On January 28 a new x-ray examination was ordered; this showed more decalcification of the bone adjoining the fracture line. The patient's general condition had improved so that she could be up and about; she gained weight and said she felt better than she had for a long time.

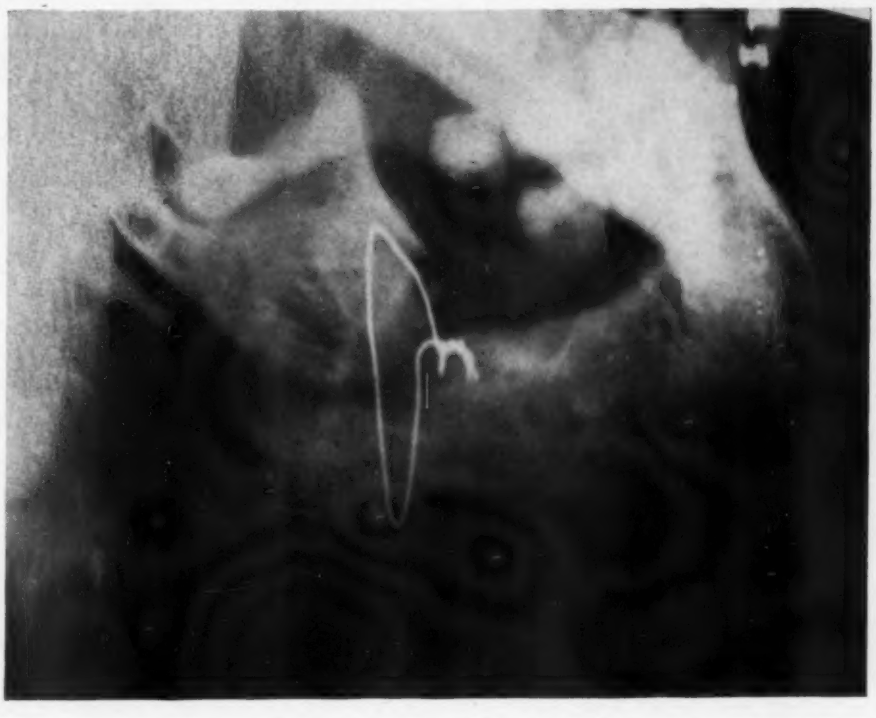

Fig. 105.—X-ray showing good callus formation at posterior end of the fracture.

On January 27 the x-ray examination for the first time showed "what appears to be the beginning of callus formation at the posterior end of the ramus where a larger area of bone had been destroyed. On January 28 the intramuscular injections of penicillin were discontinued; a total of 1,600,000 units had been administered in all. On January 31 the drain tubes could no longer be kept in the wound, and were discarded. The lower incision closed promptly; in the other a slight amount of seepage remained. Cultures from this still showed the presence of *Staphylococcus aureus* (February 1), therefore it was irrigated with penicillin the succeeding days. On February 5 another blood transfusion of 500 c.c. was given. On February 7 the fistula was dry.

On February 9 the intraoral splint was taken out to test the jaw, which was found to be solid. The patient felt no pain on manipulation. The dentures

were separated, to be worn for another week or ten days with a Barton bandage. The x-ray taken at this time showed good internal callus filling in from the posterior margin to the middle of the ramus, though there was not much healing observed in the anterior part of the fracture where the tooth had been removed (Fig. 105).

The patient was discharged on February 10. On February 16 the bandage was discarded and exercises prescribed to overcome the slight muscular spasm which was present. An x-ray taken in March showed the fracture healed completely.

Discussion.—The first lesson to be learned from this case pertains to the time when unerupted impacted third molars should be removed. I feel strongly that the time to remove them is when the patient is young, preferably before the root formation has been completed, but certainly before the third decade of life. Also, no unerupted teeth should be left when the other teeth are removed to give the patient full dentures. Invariably, such teeth cause trouble because of the pressure and friction a denture produces, and the infection which sets in. In elderly people with brittle bone and marked atrophy of the jaw, fracture occurs easily, particularly if the tooth, because it was not in function, has undergone resorptive changes which are generally followed by bony ankylosis, as in this instance.

This case also illustrates the dramatic change brought about by treatment with penicillin. Intensive local treatment combined with good supportive medication over a period of one month was of no benefit. The fracture failed to heal, and suppuration remained prolific until penicillin therapy was instituted. The discharge, which was profuse on January 3 when the treatment was started, as seen in Fig. 99, had almost completely ceased on January 11, as shown in Fig. 101. The fracture showed a good callus, so that the jaw was firm four and one-half weeks after treatment with penicillin had been started. In all, 1,600,000 units in dilution of 5,000 units per 1 c.c. were injected intramuscularly over a period of twenty-five days, and local treatment in dilution of 500 units per 1 c.c. over a period of twenty days was used in addition, the local treatment being started on the fifteenth day of intramuscular therapy and continued ten days after the latter had been discontinued. The result was very satisfactory.

Case 25

Depressed Fracture of Right Malar Bone and Compound Fracture of Right Maxilla

H. H. (No. 424928), a 30-year-old soldier, presented himself at the Emergency Ward on Oct. 25, 1943. Before admission, he had been struck on the right side of the face while playing football.

Examination showed a depression of the right zygoma with a point of tenderness. There was an area of ecchymosis around the right eye. The patient had no pain, but complained of numbness in the infraorbital region, and at the side of the nose.

Roentgen examination revealed a depressed fracture of the right malar bone, as well as fracture of the lateral and superior walls of the right antrum. There appeared to be some increased density in the antrum, which was considered due to the presence of a hematoma.

He was admitted to the House on Oct. 26, 1943, for operation. Sulfadiazine therapy was begun at once and was continued through the fourth postoperative day with a blood level maintained at 9 mg. per cent. The preoperative medication consisted of $\frac{1}{300}$ grain of scopolamine given two hours before operation, and $\frac{1}{300}$ grain of scopolamine and $\frac{1}{120}$ grain of atropine on call.

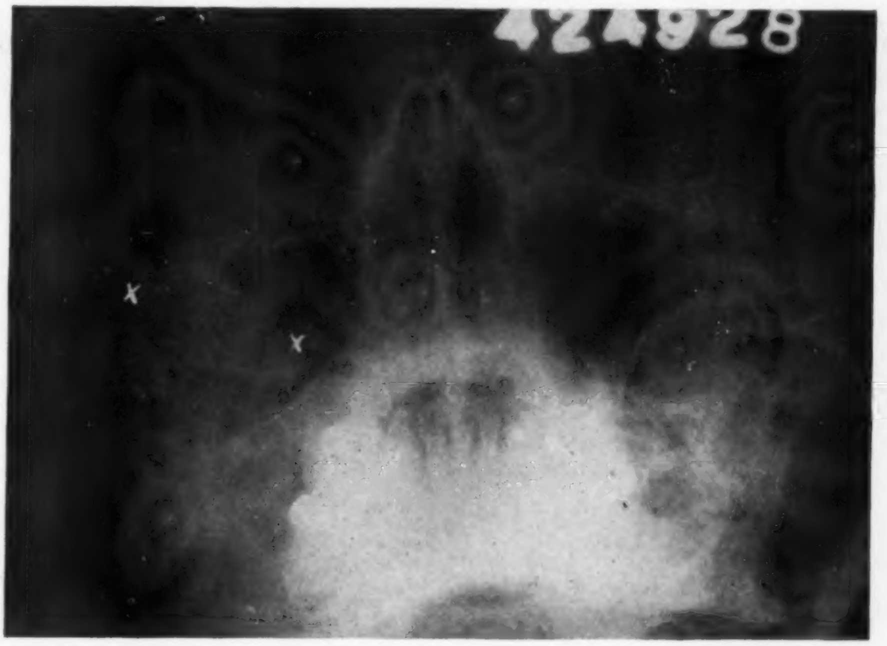

Fig. 106.—Comminuted fracture of maxilla.

An intraoral incision was made under pentothal sodium intravenous anesthesia from the median line to the molar region, about ½ cm. above the gingival margin. The mucoperiosteum was detached from the bone by means of blunt dissection, and the fracture became visible, extending vertically from the canine region to the infraorbital ridge. The maxillary sinus was opened by means of a gouge and rongeur forceps. When its contents became visible, it was found that it consisted of comminuted bone splinters and partly organized blood. One large splinter, consisting of the posterior wall of the sinus and the lower surface of the malar bone, was projecting into the sinus. It probably was the spicule of bone that gave the curious appearance in the x-ray (Fig. 106). Four pieces of bone which were detached from the periosteum were removed, after which it was found that the contents of the pterygopalatine fossa protruded into the sinus cavity. The remaining fragments, the malar bone, and the roof of the orbit were pushed back into place, after which a boric strip was inserted to fill the antrum completely. The gauze was allowed to protrude through the incision which was closed on both sides with interrupted silk sutures. Postreduction x-rays showed that the deformity had been corrected.

Progress was favorable. Chemotherapy was discontinued on November 4 and the patient was discharged on the fifth postoperative day, to be followed in the Outpatient Department.

When he was seen on Nov. 17, 1943, the wound was well healed. There was a slight retraction scar in the canine fossa. The patient still felt some numbness in his teeth, though they were neither sore nor painful, and there was still a little numbness in his upper lip. The anesthesia of the lateral side of the nose was relieved, however. He was discharged with instructions to report if he had any soreness or pain, and was further advised to have x-rays of his teeth in about two months.

On Jan. 17, 1944, the patient returned to the Outpatient Department complaining of his teeth being somewhat sore when he brushed them and when he ate. X-rays, however, showed no evidence of any infection. There was no evidence of discharge from the maxillary sinus or soreness of the incision in the canine fossa.

Discussion.—It seems incredible that so much comminution of the walls of the maxillary sinus could be caused without more displacement of the zygoma. The patient must have been hit with the toe of his opponent's football shoe, just underneath the malar bone. Ordinarily, most of the fragments in such fractures can be decompressed, but, in this case, four pieces were detached to the extent that they had to be removed.

Case 26

Fracture of Left Zygomatic Arch

A. B. (No. 418894), a 35-year-old ship fitter, came to the Emergency Ward on Sept. 6, 1943, complaining of pain in the left side of the face, and a depression of the left malar prominence.

The night before, the patient was attacked by a number of men, who struck him about the face and head with sticks and blunt objects. A friend, who was present, said the patient never lost consciousness, but the patient himself remembered nothing from the time he was struck until he found himself going home on the subway thirty minutes later. He was told that he had walked to the subway in a normal manner. The patient experienced no great pain other than headache and soreness. There was no second lapse of consciousness, drowsiness, or vomiting. He had transient nosebleeds for two days, but there was no bleeding from the ears or leakage of cerebrospinal fluid.

At 5:00 A.M. the following morning, the patient went to work, but was forced to see the company doctor because of pain. X-rays were taken, which showed a fractured zygomatic arch (Fig. 107). The patient was admitted to this hospital.

On admission, examination showed an indentation over the left zygoma, with pain and tenderness at the left temporomandibular joint. There were tiny cuts over the bridge of the nose on the left, and at the outer edge of the left eyebrow. There was tenderness lateral to the depression about midway between the outer canthus of the eye and ear. There was tenderness over the left mastoid. There were no signs of neurological significance; there was no evidence of intracranial injury.

Sulfadiazine therapy was instituted on Sept. 6, 1943. The patient received an initial dose of 2 Gm., and then 1 Gm. every four hours. This was continued for four days.

On Sept. 8, 1943, under intratracheal nitrous oxide, oxygen, and ether anesthesia, the fracture was reduced. The preoperative medication consisted of $1\frac{1}{2}$ grains of nembutal at bedtime, and $\frac{1}{6}$ grain of morphine and $\frac{1}{100}$ grain of atropine on call. An incision was made in the reflection of the mucosa underneath the zygomatic process of the maxilla. By blunt dissection, the region underneath the zygomatic arch was reached. A periosteal elevator was inserted, and by using a finger placed on the outer wall of the maxilla as a fulcrum, outward pressure was applied by means of lever action. After some manipulation, the collapsed arch was felt to snap into position, and after sulfathiazole ointment was inserted into the wound, the incision was closed with silk sutures.

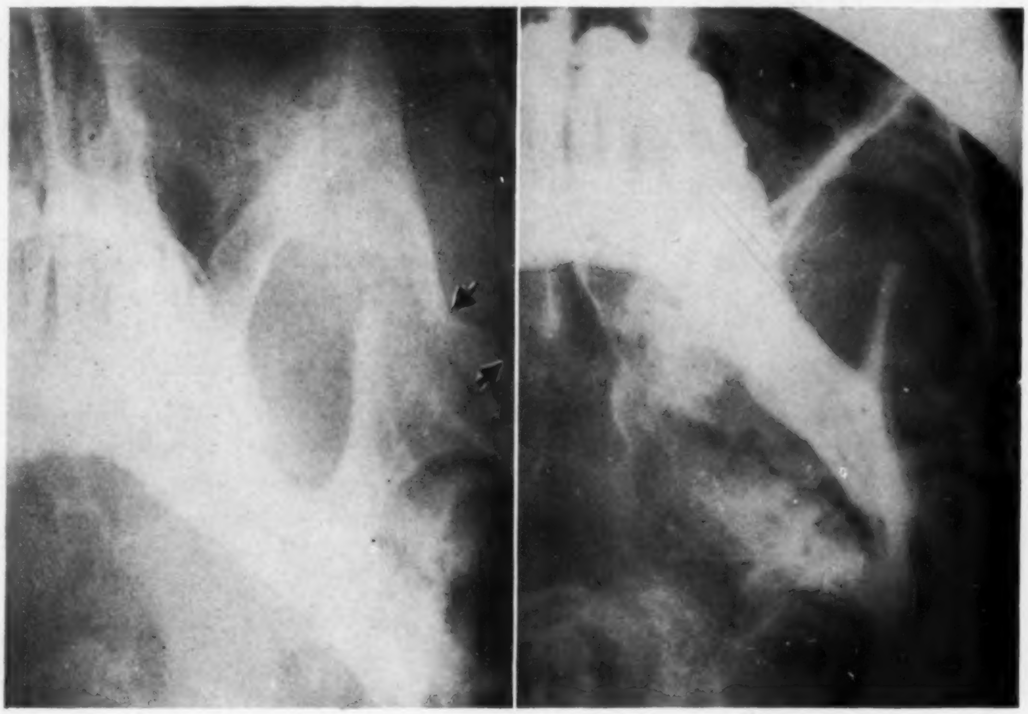

Fig. 107. Fig. 108.

Fig. 107.—Fracture of zygomatic arch.
Fig. 108.—Fracture of zygomatic arch after reduction.

Postoperative x-rays showed that the depression was corrected (Fig. 108).

The postoperative course was smooth, and the patient was discharged on Sept. 10, 1943, to be followed in the Outpatient Department.

On September 16 the patient was seen in the Outpatient Clinic. He had no swelling or complaints; the sutures were removed. The wound looked well.

Discussion.—There are several methods by which fractures of the zygomatic arch may be reduced. The one described, when used with aseptic precautions,

seems to the writer to be the simplest. Two other methods are favored by others, namely that of using a towel clamp, the points of which are pressed through the skin to grasp the fractured bone; and the other, recommended by Gillies, which consists of inserting an elevator through an incision over the temporal muscle, passing it down beneath the temporal fascia, and beneath the zygomatic arch. By careful levering with a large pad of gauze as a fulcrum, the fragments are replaced into their normal position.

Case 27

Bilateral Fracture of Mandible: Compound Fracture in Molar Region on Left, and Subcondylar Fracture of Ramus on Right

A. S. (No. 413157), a 59-year-old man, came to the Outpatient Department for the first time on July 19, 1943, complaining of pain and swelling of both sides of the jaw of six days' duration.

Six days before admission, the patient had been allegedly struck on the right side of the face during a fight, while he was at work. He was knocked to the ground but not rendered unconscious. There was considerable bleeding, which lasted through the night. Pain and swelling were noticed almost immediately on the right side, and on the following day there was swelling on the left side also. The patient had not worked since the injury and had been able to eat only soft food.

The evening after the accident, the patient saw his dentist because he thought a tooth was loose on the right side. The dentist told him that nothing could be done because of the swelling of his face. The patient treated his jaw at home with heat and with cold, but he did not experience much relief from either.

The examination revealed a foul odor coming from the mouth. There was much calcarious deposit and soft debris about the teeth. The lower right second molar protruded over the occlusal plane; it was extremely loose, and the mucosa was lacerated. There was mobility at the angle of the right jaw, and in the region of the left condyle, and crepitus when the mandible was moved. There was some ecchymosis and slight swelling on the right jaw and face. The routine physical examination disclosed mild essential hypertension, and there were a few moist crepitant râles in the right apex of the lung.

X-rays of the jaws showed a fracture through the body of the mandible on the right side, extending between the two remaining molars with upward displacement of the ramus (Fig. 109). On the left side, there was a fracture line extending through the subcondylar region of the ramus from the mandibular notch to the posterior border above the attachment of the stylomandibular ligament (Fig. 110).

Diagnosis.—Compound fracture with displacement of the right mandible; simple subcondylar fracture of the right ramus.

The patient was admitted to the House for open reduction of the fractures and intermaxillary fixation, on July 20, 1943. Sulfadiazine was given by mouth and a blood level of 7.8 mg. per cent was obtained. The operation was performed on July 23, under nitrous oxide, oxygen, and ether anesthesia. The

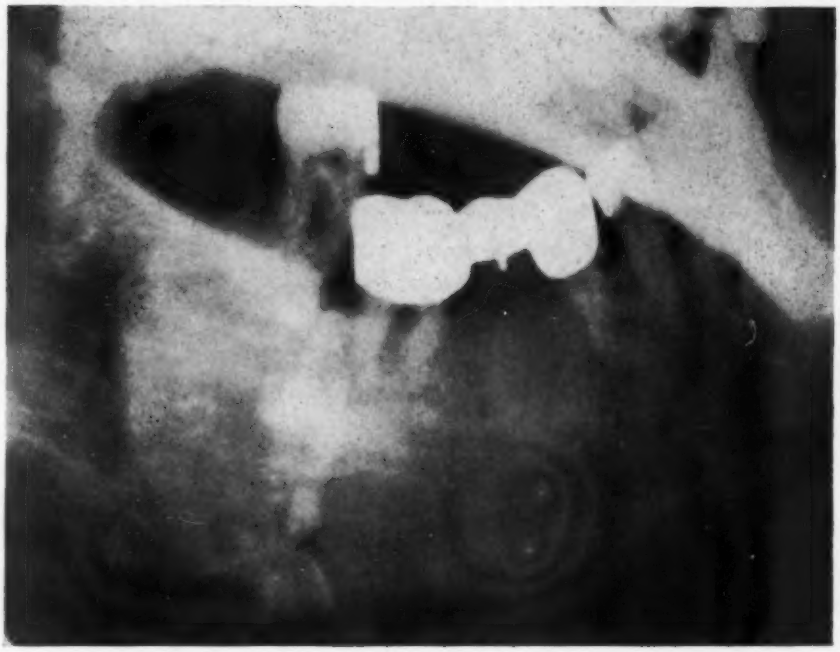

Fig. 109.—Compound fracture of right mandible with displacement of posterior fragment.

Fig. 110.—Fracture of left ramus in subcondylar region.

preoperative medication consisted of $\frac{1}{6}$ grain of morphine and $\frac{1}{100}$ grain of atropine on call. After careful preparation of the skin by the usual method, the open reduction was performed first. An incision was made below the mandible, extending from the angle of the jaw about halfway along the horizontal ramus. The external maxillary artery and vein were located and preserved by careful retraction. The mandible was exposed by blunt dissection and the periosteum incised. A hole was drilled into each fragment about $\frac{1}{2}$ cm. away from the fracture line. A stainless steel wire was drawn through the hole in the posterior fragment (Fig. 111), which was pulled down with bone forceps, then through the hole in the anterior fragment (Fig. 112). The fracture was thus reduced, and the wire twisted to hold the fragments in position (Fig. 113). The subcutaneous tissue was closed with catgut sutures and the skin with interrupted Kaldermic. Because the fracture was compounded into the mouth, the twisted steel wire was allowed to protrude through the opening for drainage and to make removal easier. After applying a dressing, the right mandibular molar was extracted. After this, Jelenko splints were attached to the upper and lower jaws, and the patient discharged to the ward to have intermaxillary elastics applied the next day when the danger of vomiting had passed. The following morning these elastics were applied and perfect occlusion obtained.

X-rays taken at this time showed good reduction of both the mandibular fracture (Fig. 114) and that of the ramus (Fig. 115).

On the first postoperative day the patient showed a slight elevation in temperature to 101° F., without an increase in respiration or pulse rate. There were signs of atelectasis with moist, atelectatic râles. On the following day the right side of the chest was clear except for some râles at the base. The atelectasis appeared to have cleared up and the temperature was approaching normal. There was evidence of suppuration at the operative site, but the drainage provided by the protruding wires seemed adequate. The patient's mouth was sprayed with the power atomizer, and the external wound irrigated with Dobell's solution. After ten days the discharge had stopped, and chemotherapy was discontinued on Aug. 1, 1943. The patient was discharged on August 2, to be followed in the Outpatient Department.

X-rays taken on Aug. 26, 1943, revealed that as yet there appeared to be no definite bony union, and that there was some resorption about the wire loop. The wire was removed on Sept. 3, 1943, through a small incision, under novocain anesthesia. X-rays taken following this procedure showed that the good position was maintained, and one week later the intermaxillary splints were removed and the mouth sprayed and cleaned.

When the patient came in on Sept. 29, 1943, there was a small fistula in the external wound, which was irrigated. By Oct. 6, 1943, the fistula was nearly closed, and there was very little or no drainage. No sequestrum could be felt.

The patient was last seen on Dec. 20, 1943. At this time there was no evidence of any fistula or infection, and the patient was discharged permanently.

Discussion.—A good result was achieved in this case by a positive method, namely, open reduction. Intermaxillary fixation was used to stabilize the

Fig. 111.

Fig. 112.

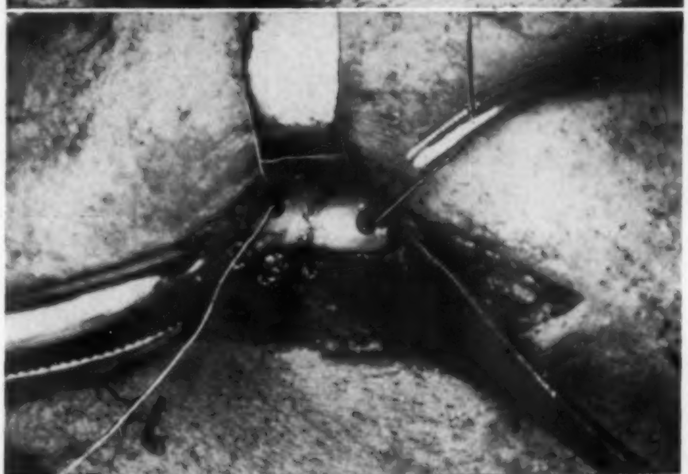

Fig. 113.

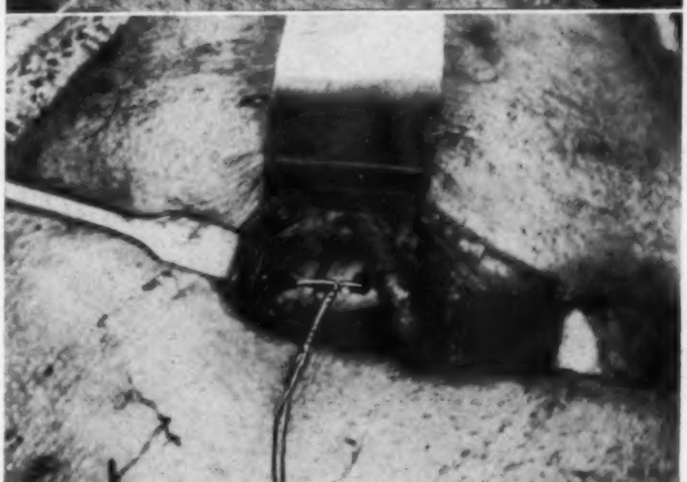

Fig. 111.—Fracture exposed. Wire inserted into hole of posterior displaced fragment.
Fig. 112.—Fracture reduced and wire inserted through hole drilled in each fragment.
Fig. 113.—After wires are twisted, reduction is complete.

Fig. 114.—Postreduction x-ray showing good position of condylar fragment.

Fig. 115.—Postreduction x-ray showing direct wiring of mandibular fracture.

condylar fracture, as well as for accessory aid to the interosseous wire. A small amount of localized osteomyelitis occurs frequently at the angle of the ramus if the fracture is compounded into the mouth, and any fracture extending through a tooth socket must be looked at as a compound fracture. However, this is of little consequence; it occurs if only one hole is drilled for a wire projecting through the skin to be attached to a bar extending from a head cap, a method which does not prevent displacement from accidental disarrangement of the appliance.

Case 28

Fracture of Ramus of Mandible

D. B. (No. 42988), a 41-year-old man, came to the Outpatient Department for the first time on Sept. 14, 1943, his chief complaint being a broken jaw.

Five weeks before admission, this patient was thrown from a wagon when the horse ran away. The wagon struck an automobile and he was thrown into the street. He was unconscious for twelve to sixteen hours following the accident. The patient was taken to a local hospital, where he was found to be suffering from a broken leg, bruises and lacerations about the head and face, and a broken jaw on the left side. He was treated for all these injuries with an improvement in all conditions except the jaw fracture, which failed to unite. The patient was referred to this Clinic for further treatment.

On examination, there appeared to be a fracture in the left mandible in the region of the first premolar, which was very loose. In the fracture line and around the tooth socket there appeared to be some infection, and there was some purulent discharge.

X-ray examination of the mouth revealed that there were two fractures of the mandible, one involving the right condyle at its base (Figs. 116 and 117), and the other, the left horizontal ramus in the region of the left lower canine tooth. There was some separation and angulation between the fragments of the condyle and the main fragment of the mandible. The fragments on the left side were also somewhat separated. The fractured surfaces appeared somewhat hazy, but they were not yet overbridged by callus formation. There was no definite evidence of osteomyelitis present, although there was some resportion of bone on the left, near the socket of the tooth where the fracture ran.

The patient was admitted to the House on Sept. 15, 1943. He was immediately placed on sulfadiazine. He was given an initial dose of 2 Gm., and 1 Gm. every four hours. The next day the fractures were reduced under intravenous pentothal sodium anesthesia. The preoperative medication consisted of $1\frac{1}{2}$ grains of nembutal at bedtime, $\frac{1}{6}$ grain of morphine, and of $\frac{1}{100}$ grain of atropine on call. Since the five-week-old fracture in the vertical ramus showed a great amount of overriding, an attempt was made to reduce it. A pin was inserted into the condylar fragment, and two pins in the angle of the jaw. One connecting bar was applied to the two pins at the angle, and a vertical bar from its posterior end to the pin in the condylar fragment (Fig. 118). This arrangement was found unstable, and another connecting bar was applied from the pin in the condyle to the anterior part of the bar at the angle, form-

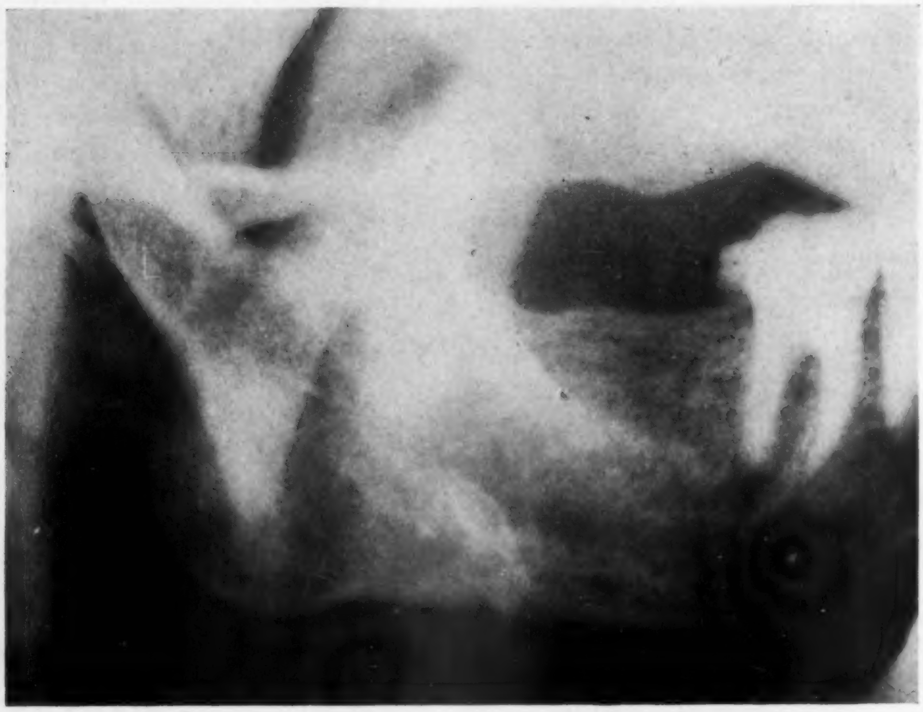

Fig. 116.—X-ray showing fracture of ramus of mandible.

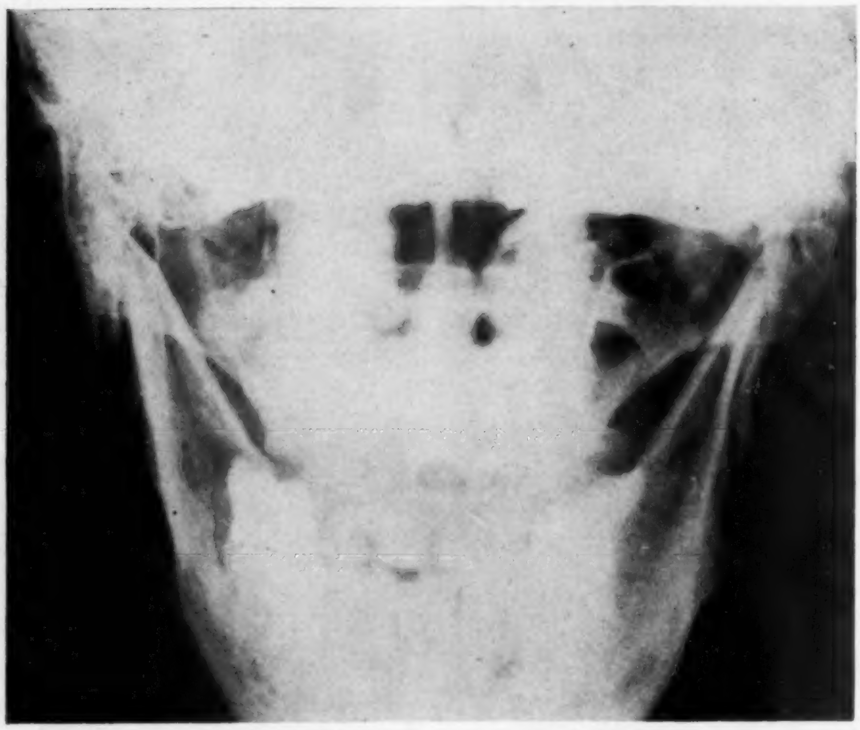

Fig. 117.—Anteroposterior view showing fracture of ramus of mandible.

ing a triangle, which gives the greatest amount of stabilization to the fractured parts (Figs. 119 and 120). Upon trying to reduce the fracture, however, by increasing the distance between the upper and lower pins, it was found that the two parts had already united somewhat, by formation of callus between the fragments, and the procedure was only partly successful. The fracture on the left side also had united fairly well, and the alignment being satisfactory, it was felt that intermaxillary ligation would suffice to stabilize the jaw until complete union should occur. The intermaxillary fixation was accomplished by wiring the patient's artificial denture to the lower teeth and holding it in place by means of a four-tailed bandage.

Sulfadiazine therapy was continued until Sept. 20, 1943. Postoperative x-rays, taken on September 18, revealed that there was still some slight angulation, but with good contact between the fragments.

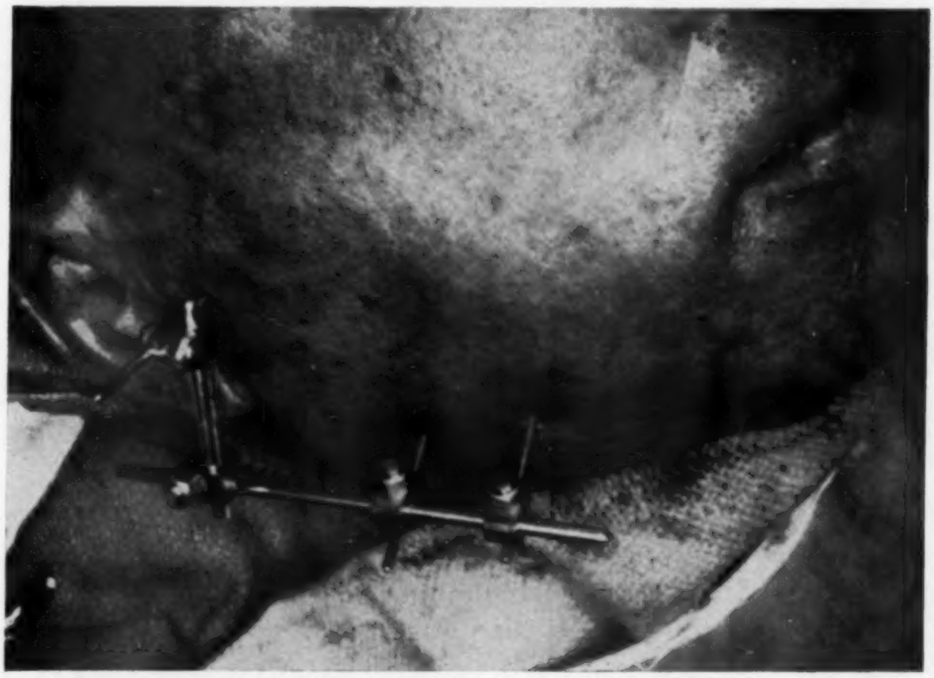

Fig. 118.—One pin inserted into the condyle is connected to posterior end of connecting bar fastened to the two pins in the angle of the jaw.

The postoperative recovery was good, and the patient, wearing a Barton bandage, was discharged on the sixth postoperative day, to be followed in the Outpatient Department.

He was again seen in the Outpatient Clinic on Sept. 27, 1943. At that time there was no discharge from the pins inserted in the ramus. The intermaxillary fixation was satisfactory, and because the fracture had been partly healed when skeletal fixation was applied, it was felt that the appliance did not contribute much to the treatment and it was removed. The pins were found firm within the bone. The mandibular fracture showed no signs of sepsis, and callus formation was evident.

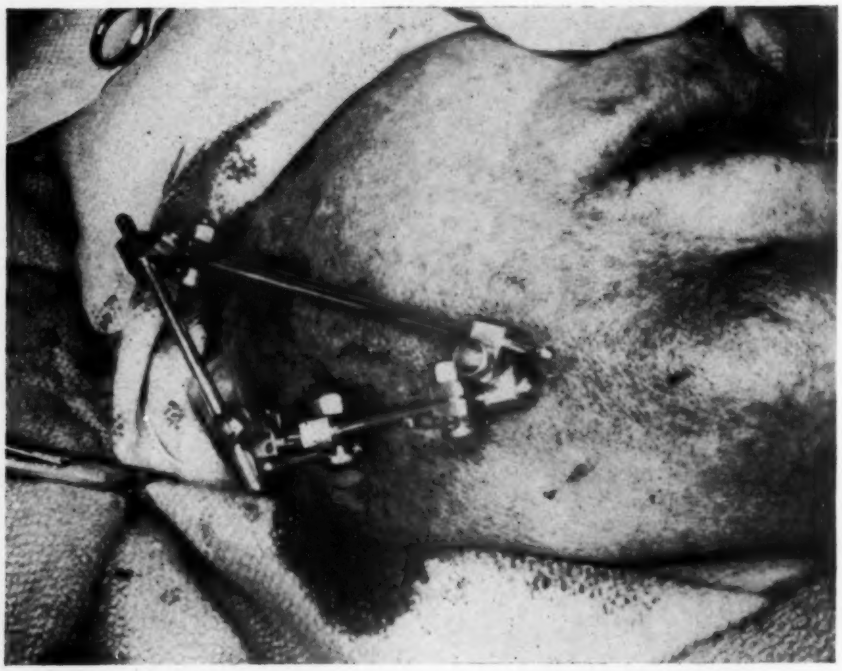

Fig. 119.—Triangle formed by means of the connecting bars to give better stability.

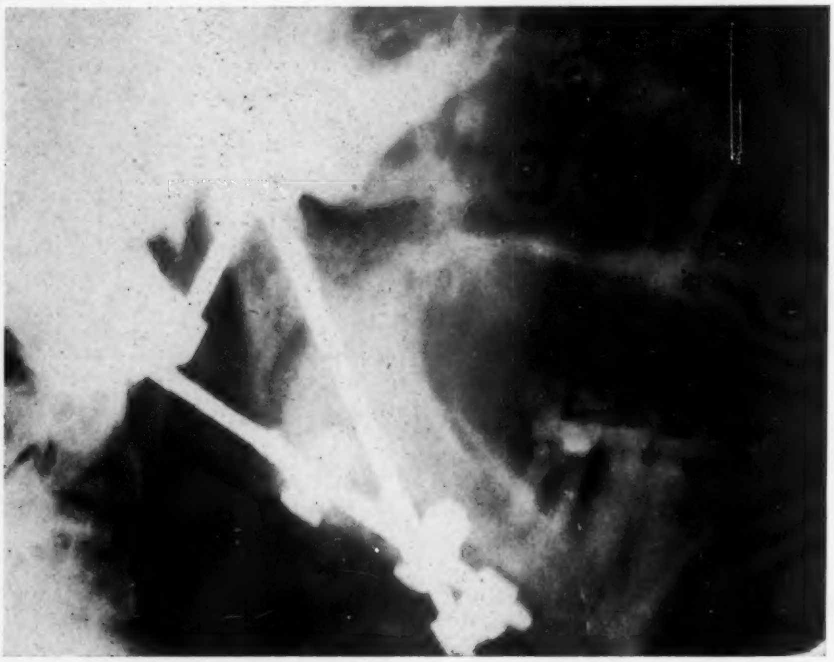

Fig. 120.—Postreduction x-ray.

On Oct. 11, 1943, the wires were removed and the patient was permanently discharged. There was considerable callus formation at the site of the mandibular fracture, and there was union of the condylar fracture. The patient was comfortable and had good occlusion of the teeth. An x-ray study of the pins made on the same day showed that the pin holes were not apparent.

Discussion.—Fractures of the vertical ramus, including subcondylar fractures, are difficult to reduce so that there is no displacement or overriding of the fragments. In the case presented, an effort was made to develop a satisfactory method of reduction and stabilization for such cases. While the result was not perfect, inasmuch as normal anatomic conditions could not be restored, the method served to give good fixation and excellent union. The resulting shortening of the ramus can be corrected easily in this patient by means of a new denture, which is needed anyway since some of the teeth have been broken.

Case 29

Subcondylar Fracture of the Mandible

H. C. (No. 421493), a 12-year-old girl, was referred to the Outpatient Department on Sept. 21, 1943, with the complaint of a fractured jaw and soreness of the teeth in the lower left canine region.

Three weeks before, the patient had been in an automobile accident and had struck her face against the dashboard. Three upper incisors were knocked out, followed by some hemorrhage. There was considerable pain of the jaw, but no unconsciousness or other injuries.

The patient was taken to a hospital in Maine, where for one week she was treated by a cap and chin strap. She then returned to her home and was treated at a local hospital, where an attempt was made to wire the teeth together. The wiring was unsuccessful, and the patient was referred to the Massachusetts General Hospital.

X-rays taken on admission showed a mandibular fracture in the left canine region and the neck of the right condyle (Fig. 121).

When admitted to the hospital, the patient had very little pain, only a soreness when she chewed on the left. The examination showed that all the jaw movements were limited. There was a palpable bony swelling along the left mandible in the canine region, which appeared to represent a callus formation and the beginning of good union. Three left upper incisors were missing and there was a little yellow fibrin in one of the sockets. Several molars were absent. Mild caries and some periodontal infection were noted.

On Sept. 24, 1943, a reduction of the condylar fracture was attempted under endotracheal ether anesthesia. The preoperative medication was $\frac{1}{12}$ grain of morphine and $\frac{1}{150}$ grain of atropine on call. Since the condylar fracture showed medial displacement and overriding, it was decided to use reduction by traction.

A half pin was inserted from the surface of the skin in front of the tragus of the ear into the condylar fragment (Fig. 122), and two pins were inserted into the mental prominence of the mandible, and connected with a crossbar. Two Jelenko splints were attached, one to the upper and one to the lower jaw.

The latter served to immobilize the partly healed fracture in the horizontal ramus of the mandible; the two together were to serve later for intermaxillary ligation by means of elastic bands.

A bite block, which was to serve as a fulcrum, was inserted in the molar region on the side of the condylar fracture, and attached to the teeth. The patient had previously had a plaster cap made with rods extending from the side and in front. The front rod was used to apply elastic traction by means of an elastic band to the pins inserted in the chin, which was to pull the mandible up and the ramus down. The pin in the condyle was connected with the lateral rod in the plaster cap by means of an elastic band (Fig. 123), to pull the condyle up and hold it in the glenoid fossa, tilting it so as to make contact with the ramus. In spite of this arrangement the overriding was not completely overcome, and the bite block was removed and intermaxillary elastic traction was reapplied on the Jelenko splints. Since the reduction of the fractures, the patient had been comfortable and without pain. An x-ray taken at this time and reproduced in Fig. 124 shows the fragments in contact, but the overriding not eliminated.

On Oct. 8, 1943, the fracture appliance was revised under gas, oxygen, and ether anesthesia. The preoperative medication was $\frac{1}{12}$ grain of morphine, and $\frac{1}{150}$ grain of atropine. The pins inserted into the chin were removed, and an attempt was made to reduce the condylar fracture by manipulation, holding the pin in the condyle with one hand and the mandible with the other. Probably because the fracture was five weeks old, it was impossible to improve the position of the fragments very much. They could not be impacted against each other. Therefore, the elastics were again applied to the Jelenko splints to achieve normal occlusion and intermaxillary fixation. The half pin in the condyle was used to hold the latter in the glenoid fossa by means of an extension rod from the plaster cap (Fig. 125).

On the following day, the wound of the pin in the right condyle was well closed, and there was no discharge. The x-ray showed the teeth to be in good occlusion, and the patient was discharged from the House to be followed in the Outpatient Department.

On Oct. 13, 1943, the patient was seen in the Outpatient Department. The elastics were found ineffective as she had been trying to open the jaw. Therefore, the fixation was improved by intermaxillary wiring. The pin in the condyle was removed and x-rays were taken. These showed no change in the position of the fragments.

On Nov. 17, 1943, all the fixation was removed. The patient had reasonably good motion of the mandible and excellent function of the condyle. The gingivae were somewhat inflamed and hypertrophied. Advice was given regarding the brushing of the teeth and the home care of the mouth.

When the patient was seen on Nov. 29, 1943, she had very good function of the mandible. On palpation, the right condyle moved normally and she could open her jaw the normal distance without any trouble. There was no pain or discomfort on mastication. Also, there were no visible scars, either on the chin or where the pin was inserted in the condyle.

The x-rays taken on November 29 revealed that the fracture had healed in excellent position, leaving no deformity. The fractures could barely be made out at that time (Fig. 126).

Fig. 121.—X-ray showing fracture of the neck of the condyle of mandible.

Fig. 122.—X-ray showing pin inserted into the neck of the condyle of mandible before traction was applied from head cap. Note overriding and lack of contact.

Discussion.—An experiment is presented which was undertaken to improve the treatment of condylar fractures. We can deduce from it that the use of a large fulcrum in the molar region does not contribute to a consequent reduction of the overriding of the fragments. This can be easily understood on

Fig. 123.—Large bite block in molar region and elastic traction were used to overcome overriding of condylar fragments. The pin in the condyle is attached to a rod in the plaster cap with an elastic band, to pull the condyle into the glenoid fossa.

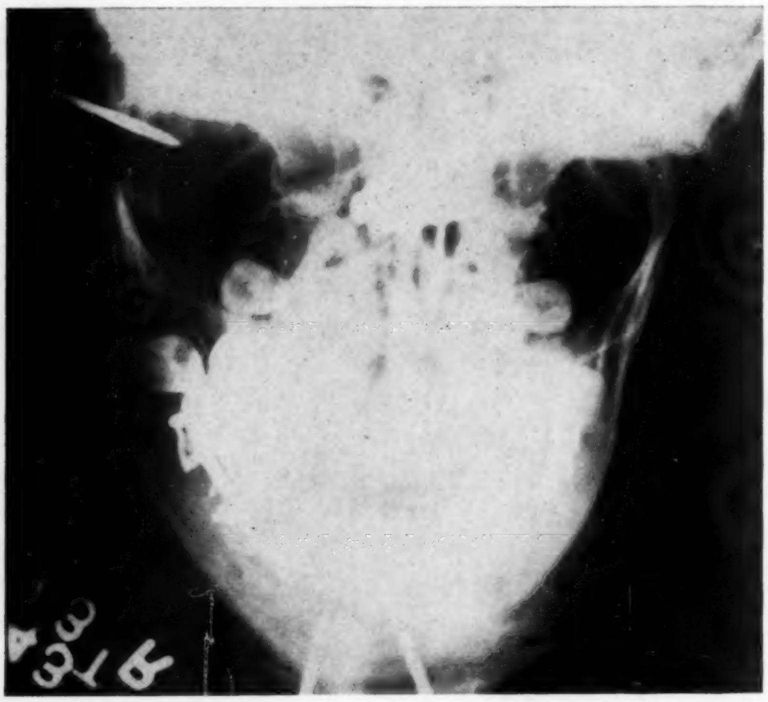

Fig. 124.—Fracture through the neck of the condyle. Pin inserted to hold the condylar fragment in contact with the ramus. In spite of traction applied to the chin where two pins had been inserted, overriding could not be overcome. However, there is good contact of the fragments.

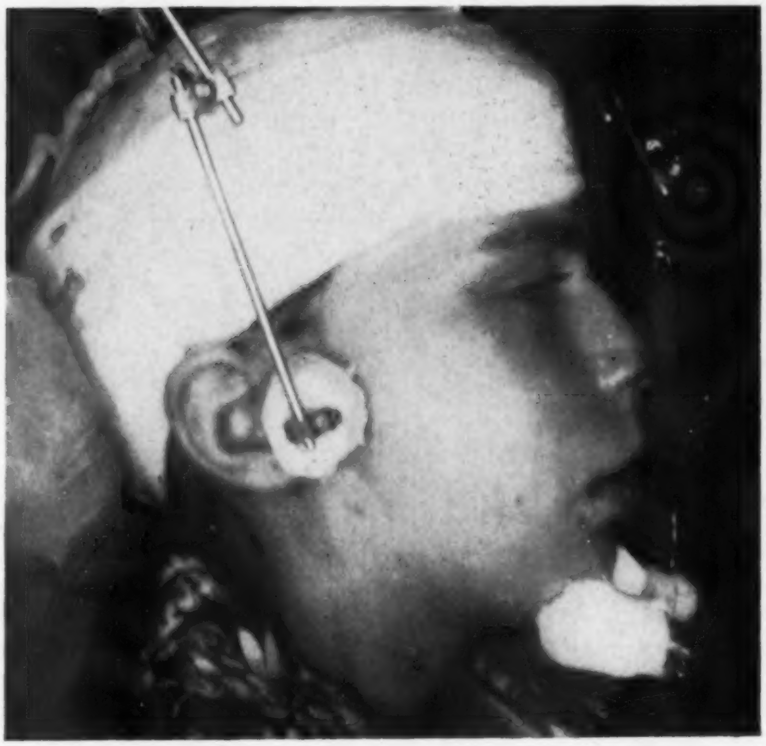

Fig. 125.—The pin in the condyle is fastened to a rod inserted into the plaster cap by means of Frac-Sure links and a bar.

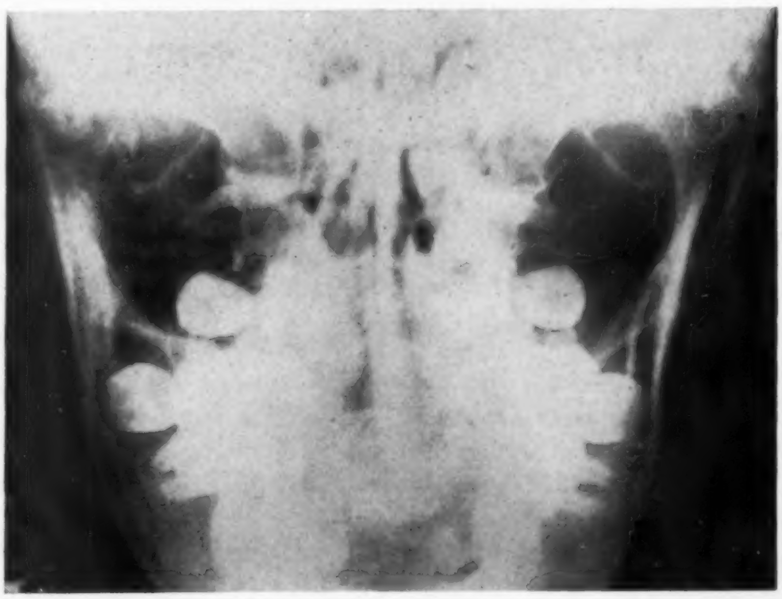

Fig. 126.—X-ray showing complete union, but slight thickening of the neck of the condyle.

considering that there is a definite limitation to the downward pull of the ramus, because of the inelastic stylomandibular and sphenomandibular ligaments which hold the jaw in suspension.

It was then thought that pulling the condyle up into the glenoid cavity might solve the problem. This procedure, however, although permitting the positioning of the condyle and bringing it into contact with the ramus, did not eliminate the overriding. In spite of this, the end result was excellent, from the point of view both of occlusion and of function of the joint.

Case 30

Multiple Face Fractures

R. C. (428844), a 34-year-old woman, was admitted to the hospital on Nov. 27, 1943, with a complaint of a painful, swollen face.

One week before, the patient was in an automobile accident. She did not remember details, but apparently the right side of her face struck the windshield, and she was unconscious for one-half hour. Her face sustained lacerations around the eye and at the angle of the mouth on the right, which were sutured at an outside hospital where she was in the care of an orthopedic surgeon who treated her for shock and brain injury. She had hemorrhage from the left ear, was unable to swallow, and her neck was sore. X-rays, taken shortly after the accident, revealed no skull fracture but multiple fractures of the upper and lower jaws. The headache which she had had following the injury disappeared, and she had no further loss of consciousness. Hearing in the left ear was lost at the time of the injury but has since been restored. There was a discharge from the nose, but this was hemorrhagic in character and not clear as in cerebrospinal rhinorrhea. X-rays, it was stated, revealed no skull fracture, but fractures of the jaw were reported. When the patient was seen in consultation, it was found that she had marked swelling and ecchymosis of the face and neck and was unable to move the jaw or to swallow. She had no ophthalmologic signs that could be detected by direct examination; neurological signs had subsided, except numbness in the lower lip on the right. The x-rays that had been taken were reviewed; these were of poor quality, but it was possible to detect a fracture of the right horizontal ramus and the left condyle of the mandible, a maxillary sinus on the right which was radiopaque, and a possible fracture of the right malar bone. The patient was transferred to this hospital for a more careful evaluation of her injuries and treatment.

On examination it was found that her general health and strength were good. The system review was essentially negative. The right side of the face was swollen and ecchymotic. There were well-healed lacerations at the angle of the jaw and under the right eye. The conjunctiva of the right eye was injected (Fig. 127). The deformity of the lower jaw on the right was apparent on looking into her edentulous mouth. There was a large hematoma present, obliterating completely the mucobuccal fold from the third molar region to the corner of the lip. There was evidence of old hemorrhage in the left ear, and some oozing of blood from the right nostril.

X-rays taken on the day of entry revealed a comminuted fracture through the right mandible, about 4 cm. from the angle, with upward and medial dis-

placement of the central fragment (Fig. 128). A 3 cm. splinter of bone lay between the separated fragments of the lower margin. A second fracture of the mandible on the left side extended through the ramus. There was lateral displacement of the central fragment. The condylar process was separated from the ramus, dislocated, and displaced. It was dislocated anteriorly and down from the condylar fossa (Fig. 129). This fracture was better visualized in a later film shown in Fig. 136, which demonstrates not only the subcondylar fracture (*C*) but also a fracture of the coronoid process of the jaw (*A*). No fractures of the skull were visible. The petrous ridges were smooth, although there may have been a fracture through the tympanic plate causing the bleeding from the ear which could not be seen in the roentgen film. The posteroanterior film showed a fracture of the wall of the maxillary sinus on the right, without much displacement, but with evidence of increased density believed to be due to hemorrhage. The findings were those of comminuted fractures of the right mandible, with fracture and dislocation of the left coronoid process and mandibular articulation, and fracture of the right maxilla.

Fig. 127.—Multiple fractures of face with swelling and ecchymosis of the face and neck.

Sulfadiazine therapy was instituted and the patient received 1 Gm. every six hours. On November 29, under nitrous oxide, oxygen, and ether anesthesia, an open reduction of the fractured mandible and nasoantral drainage of the right maxillary sinus was performed. The preoperative medication consisted of $\frac{1}{300}$ grain of scopolamine, two hours before operation, $\frac{1}{300}$ grain of scopolamine, $\frac{1}{6}$ grain of morphine, and $\frac{1}{150}$ grain of atropine one hour before operation. An incision was made intraorally, extending along the entire ridge of the right side of the mandible. A large hematoma was evacuated; some of the partly organized hemorrhage had the consistency of a rubber sponge. A very badly

displaced fracture was next located. The two main fragments were exposed and a hole drilled in each side. The fracture was reduced, but a piece was found missing; it was lying loose in the wound and had to be removed. The fragments were approximated as well as possible, and fixed by means of a stainless steel

Fig. 128.—X-ray showing comminuted fracture of mandible which contained a large hematoma.

Fig. 129.—Anteroposterior view showing fractured condyle dislocated and displaced anteriorly and downward. Fractured condyle, C_1, normal condyle, C_2, and mastoid process, *M*.

wire drawn through the holes. Because it was an oblique fracture, a circumferential wire was inserted and fastened around two fragments, as well as another long spicule at the inferior border of the mandible. This could not be placed into perfect position and was left to be dealt with later, if necessary, by

a supplementary procedure. Sulfanilamide powder, 2 Gm., was dusted into the wound, which was closed with a continuous silk suture after inserting a rubber dam drain to prevent a hematoma from reforming. A trocar was inserted into the right nostril to perforate the nasoantral wall. The hole was enlarged to provide good drainage after aspirating the accumulated blood and secretions.

Postoperative x-rays, taken on November 30, showed the fracture reduced and the fragments held in place with double wire loop (Fig. 130), without any evidence of displacement except for a linear fragment previously described lying beneath the major fragments. Additional anteroposterior views showed the fragments in satisfactory alignment in that projection (Fig. 135). The loose splinter of bone beneath the major fragment was held within the wire loop and extended across the fracture line. The fracture of the neck of the left mandibular condyle still showed medial dislocation and lateral displacement of the distal fragment, and overriding of about 1 cm.

Fig. 130.—Postreduction x-ray of mandible showing internal and circumferential wiring.

The patient made an excellent recovery from the operation, having no pain or nausea and only slight swelling of the face. The sulfadiazine therapy was continued, and a high-protein diet with vitamins was prescribed.

The rubber dam drain was removed on the third day, and the wound was found to be clean. The swelling was gradually reduced by means of cold applications. The sulfadiazine level of the blood was 10 mg. per cent on December 3.

Dec. 3, 1943, the patient was well enough for the second operation for reduction of the dislocated and displaced condyle fracture, under pentothal sodium intravenous anesthesia. The preoperative medication consisted of $1\frac{1}{2}$ grains of nembutal at bedtime, 2 grains of luminal sodium and $\frac{1}{300}$ grain of

scopolamine two hours before operation, and $\frac{1}{300}$ grain of scopolamine, $\frac{1}{150}$ grain of atropine and $\frac{1}{6}$ grain of morphine one hour before operation. After the usual preparation of the shaved temporal region and skin anterior to the ear, the latter being plugged with cotton, an angulated vertical incision was made in front of the ear, extending as far as the attachment of the lobe of the ear to the face. The tissue was divided and the transverse facial artery and vein were located, cut, and ligated. The subcutaneous tissue was then dissected, the zygomatic arch located and exposed. When the glenoid fossa was found, the meniscus was still partly attached, but the condyle was completely dislocated. It was located in the anterior and medial position indicated in the x-ray. After further blunt dissection it could be grasped with bone forceps and elevated into a position which allowed a hole to be drilled just below the condylar head. A half pin was inserted, by which the condyle could be manipulated (Fig. 131). The dislocation then was completely reduced, and the assistant, manipulating the mandible under the drapings, helped impact the fragment against the ramus. Next the subcutaneous tissue was closed with catgut sutures, and the skin with a subcuticular suture. The pin, which extended through the incision (Fig. 132), was to serve as a drain for oozing blood and to immobilize the fractured part by means of a metal bar which was attached to a Woodard appliance previously placed on the patient's head (Fig. 133). The next step was to immobilize the mandible. Since the patient was edentulous and had lost her dentures when the accident occurred, this was accomplished by inserting a half pin on each side of the chin, and attaching these by means of Frac-Sure links to the extension rods from the headband previously described (Fig. 134). This appliance, therefore, served to hold the condyle in position and to prevent motion of the mandible. Sulfadiazine therapy was continued, 1 Gm. every six hours, until December 15. On December 8 the sulfadiazine blood level was 6.6 mg. per cent.

X-rays taken on December 6 showed the position of the fragments of the mandibular fracture on the right maintained. On the left they showed that the overriding of the condylar process had been corrected, but there was still slight lateral displacement of the mandible in relation to it (Fig. 135). Another film showed that the coronoid process was fractured from the mandible on the left, and that the fragment to which the temporal muscle was attached, was displaced medially and anteriorly (Fig. 136).

The postoperative course was quite uneventful except for some bleeding from the nose, which occurred when applying pressure over the right canine fossa where a swelling had developed. On the seventh postoperative day the swelling of the face was becoming less, but the headband was uncomfortable. There was minimal edema of the scalp. Since the rectal temperature was 100° F., the sulfadiazine therapy was continued, and an adrenalin pack was placed into the inferior meatus of the nose on the right. The next day, December 7, the pain and swelling over the right maxillary sinus had become very severe, and the patient had to be given codeine and aspirin, p.r.n., during the day, and $1\frac{1}{2}$ to 3 grains of nembutal at night. New x-rays were taken, which again called attention to the fracture of the outer wall of the right maxillary sinus.

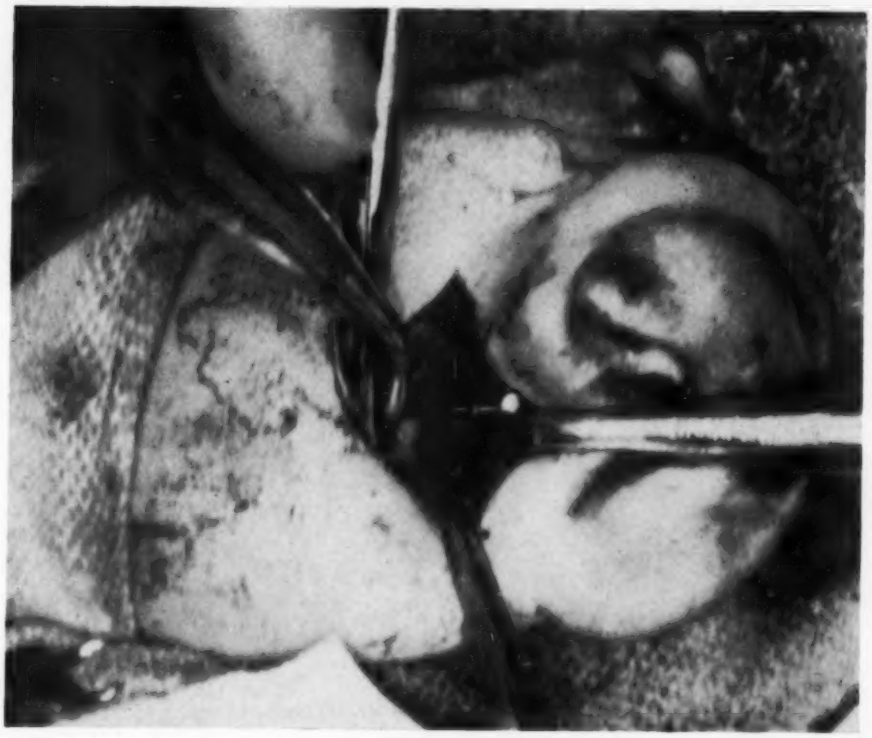

Fig. 131.—Open reduction of fractured condyle. Half pin inserted into subcondylar area.

Fig. 132.—Open reduction of condyle. Skin closed with subcuticular suture allowing pin to project through the incision.

Fig. 133.—Photograph taken three weeks after the insertion of the pin into the condyle. Suture had been removed on the tenth day.

Fig. 134.—Woodard headband applied, with two rods extending to pins in chin to immobilize the mandible, and one rod attached to the pin in the condyle.

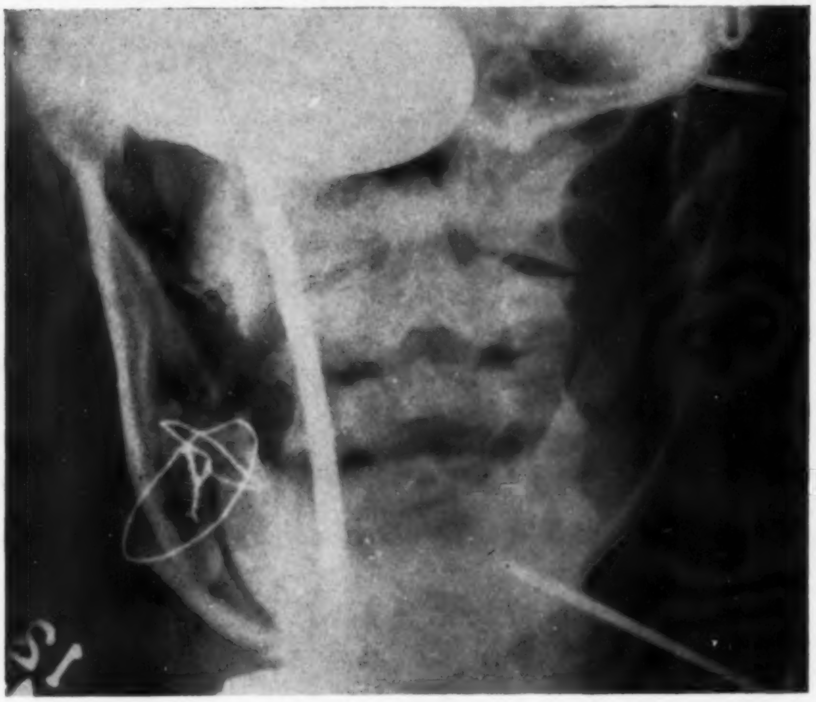

Fig. 135.—Postreduction x-ray showing pin inserted into subcondylar area as well as wiring of comminuted fracture of mandible. Note pins inserted into chin, which were attached by rods to the headband (see Fig. 134).

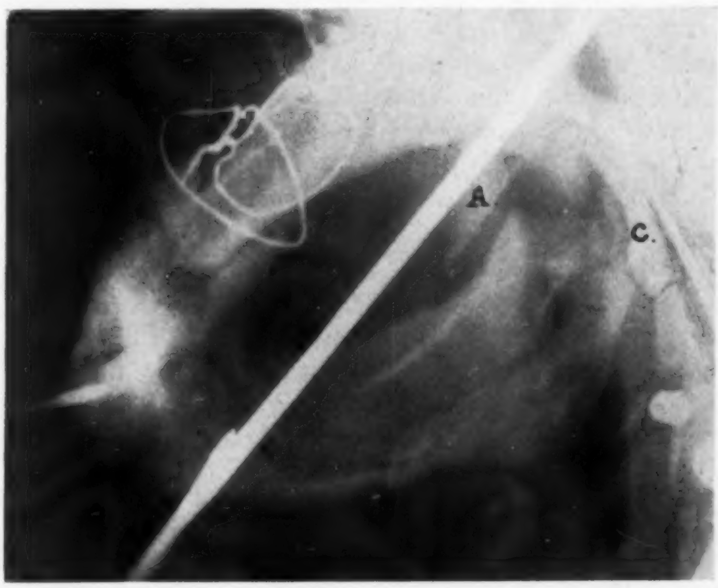

Fig. 136.—Postreduction x-ray shows subcondylar fracture, *C*, reduced, and fracture of coronoid process of ramus on left, *A*, unreduced.

On December 12 a hematoma was visible under the upper lip; it seemed to extend over the outer wall of the maxilla, and it was assumed that this might have been caused by a slow hemorrhage in the sinus which at the same time caused the nosebleeds.

On December 14 under pentothal sodium anesthesia with the usual premedication, an intratracheal tube was inserted and the nasopharynx packed. An incision was made in the right canine fossa: this entered a hematoma, and a considerable amount of clotted blood was evacuated. The wall of the maxillary sinus was found to be fractured. Part of it was removed, and it was discovered that the cavity was filled with partly organized blood clots. After removing the hemorrhagic mass in the sinus, including several pieces of detached bone, a very severe hemorrhage ensued, blood pouring out of both the nostril and the wound. The blood was taken up by the aspirator, and the hemorrhage was promptly stopped with adrenalin packs inserted into the sinus and into the nose. The adrenalin packs were removed in order to find the bleeding vessel, presumably the infraorbital or superior alveolar artery. The hemorrhage, however, seemed to be associated with the fracture of the posterior wall, which, when manipulated; caused renewed bleeding. Therefore, it was deemed wise to pack the sinus with a boric strip, which, through pressure, insured hemostasis. It was allowed to protrude through the incision. The pathologic examination of the antral contents showed blood clot and acute inflammatory exudate.

The following day the patient felt weak and was rather "washed out." A blood transfusion was advised but was refused by the patient. On December 18 she felt very much better, particularly because the nosebleeds, which had worried her a good deal, had stopped and the pain had disappeared.

On December 20, under gas and oxygen anesthesia, the pack was removed in the operating room; all precautions were taken, such as a pack in the nasopharynx, suction in readiness, and adrenalin at hand. Hydrogen peroxide was injected into the dressing with a hypodermic needle; this loosened the antrum pack so that it could be removed easily. No bleeding occurred and a much smaller boric strip was inserted to keep the wound open.

The patient continued to do well. The dressing was changed every other day. She was discharged from the hospital on December 23, and seen for dressings every three or four days. The pin in the condyle was removed on Jan. 7, 1944. On January 10 the antrum had stopped draining. An x-ray taken of the fracture of the mandible was satisfactory, but there was some swelling in front of the left ear although the pin wound had completely healed.

On Jan. 14, 1944, the patient was again admitted to the hospital. Under gas, oxygen, and ether anesthesia, the maxillary sinus was closed by sutures, the pins were removed from the chin, and the headband discarded. The mandibular fracture had apparently healed. The swelling over the left condyle was explored, and, after making a stab incision at the place where the pin was formerly inserted, a slight amount of pus escaped. On culture this yielded *Staphylococcus aureus.*

The following day the fistula was treated every five or six hours with zephiran injected into the wound. It was now possible to take good x-rays,

which was impossible before because of the external apparatus applied for fixation. The condyle appeared to have been somewhat displaced; this was attributed to the headband, which is likely to become displaced when the patient tosses in bed at night. The question arose as to whether the original displacement of the condyle had destroyed its blood supply, or whether it had become infected when the patient had the acute inflammatory condition in the maxillary sinus. A divided Gunning splint was constructed, inserted, and supported with a Barton bandage reinforced by elastic traction. Since I had some diluted penicillin left from another patient, the fistula was somewhat enlarged and treated by injecting penicillin, 500 units per 1 c.c., deep into the wound several times a day. This treatment was started on January 31 and continued until February 17, when the discharge had been arrested. The fistula closed spontaneously. The patient was discharged from the hospital on the following day, but was carefully followed up. When she was seen on March 15, the x-ray showed good callus between both the coronoid and condyloid processes and the ramus, and although there was some muscular trismus present, the joint seemed to function satisfactorily. On March 20 she was ready to have an impression taken to have new dentures made. Since the circumferential wire could be felt under the alveolar mucosa, it was thought best to remove it to prevent irritation.

Discussion.—The patient presented the type of multiple injuries which requires a good deal of planning to obtain a satisfactory result. The fact that no complication occurred in the mandibular fracture is more or less a triumph for internal wiring fixation. The condylar fracture also gave an unexpected good result. A dislocation fracture with the condyle displaced as much as was the case in this patient, is generally believed to have a poor prognosis, because, no doubt, the condylar blood supply in such a fracture must be completely severed. When infection set in, I certainly felt that there was little hope, and had it not been for penicillin, I would have performed a condylectomy at once. The result shows what patience and perseverance will sometimes accomplish.

Case 31

Multiple Fractures of the Face and Base of the Skull

G. C. (No. 426469), a 38-year-old woman, was brought to the Emergency Ward on Nov. 9, 1943, twenty-one hours after an automobile accident. She was suffering from multiple facial fractures, lacerations which had been sutured, and bleeding from the mouth and nose.

The patient had apparently been thrown against the windshield of the automobile. Allegedly, she was not knocked unconscious. Her local doctor said that she had not been drowsy, that she had been nauseated and had vomited several times, and that many teeth had fallen out of the upper jaw. She had taken small amounts of fluid by mouth. At no time did the patient have convulsions, motor or sensory symptoms.

Examination revealed a markedly swollen face. There was periorbital ecchymosis. The left eye was closed. Both pupils were enlarged and fixed, with the left pupil irregular and larger than the right. The patient was blind

in the left eye, and there was paralysis of the sixth nerve on the left. There was a massive hemorrhage in the left fundus which, it was felt, might be causing a detachment of the retina. Otherwise there was no motor or sensory loss. The reflexes were equal and active. There was a tenseness and swelling over both mandibles and maxillas, especially over the right maxilla. The teeth were loose, and several were absent. The patient could open her mouth very little. The several lacerations about the lips and chin had been sutured. There was some bleeding from the nose and cerebrospinal rhinorrhea from the left nostril (Fig. 137). The nasal bones were prominent, with the cartilages depressed. There was no apparent trauma to the skull or scalp. There was no stiffness in the neck.

The patient was instructed to rest in bed, and an intravenous infusion of 500 c.c. saline with 5 Gm. sodium sulfadiazine was started, and then 1,500 c.c. of 10 per cent dextrose and water were given.

X-rays of the skull and jaws, taken on Nov. 10, 1943, revealed a fracture through the base of the skull involving the cribriform plate. In the lateral view of the head, the entire facial skeleton was seen detached from the base (Fig. 138). The same x-ray shows a transverse fracture of the maxilla. There was no fracture of the vault. The horizontal ramus of the right side of the mandible was fractured in the region of the canine (Fig. 139), and probable fracture of the neck of the mandible on the left was seen. There were multiple fractures of the bones of the middle third of the face, including the nasal bones, the maxillas, and both malar bones. The malar bone on the left and the orbit showed severely comminuted fractures (Fig. 143, white arrows). There was a large amount of soft tissue swelling posteriorly in the nasopharynx, which could be due to the fracture of the base. Displacement of the facial bones in a posterior direction is well shown in Fig. 143, in which the black arrows indicate the amount of retrusion.

These fractures, it was agreed, should not be treated until the patient was over the shock and the fracture of the cribriform plate had a chance to begin to heal.

On Nov. 11, 1943, the eye consultant said that there was no separation of the retina, and that the prognosis for return of vision was good. On November 12 the patient's temperature spiked to 102.5° F. She received 2½ Gm. sodium sulfadiazine intravenously, and also intravenous vitamin therapy which consisted of 4 mg. menadione bisulfite, 10 mg. thiamine hydrochloride, 10 mg. riboflavin, 500 mg. ascorbic acid dissolved with 50 Gm. amino acid, and 1,000 c.c. of 5 per cent dextrose with 8.96 Gm. sodium racemic lactate.

On November 13, a rubber dam drain was inserted in the fracture site through the laceration, after removing a suture, because a fluctuant swelling had formed. Chemical tests that were made the day before were reported as follows: sulfadiazine level in the blood, 4.3 mg. per cent; chloride, 93.4; and nonprotein nitrogen, 20. Chemical tests made on November 15 showed the sulfadiazine level in the blood to be 6.6 mg. per cent, and protein to be 6 mg. per cent.

Fig. 137.—Patient sustaining middle third facial and mandibular fractures from auto accident. Note cerebrospinal fluid running from the nose.

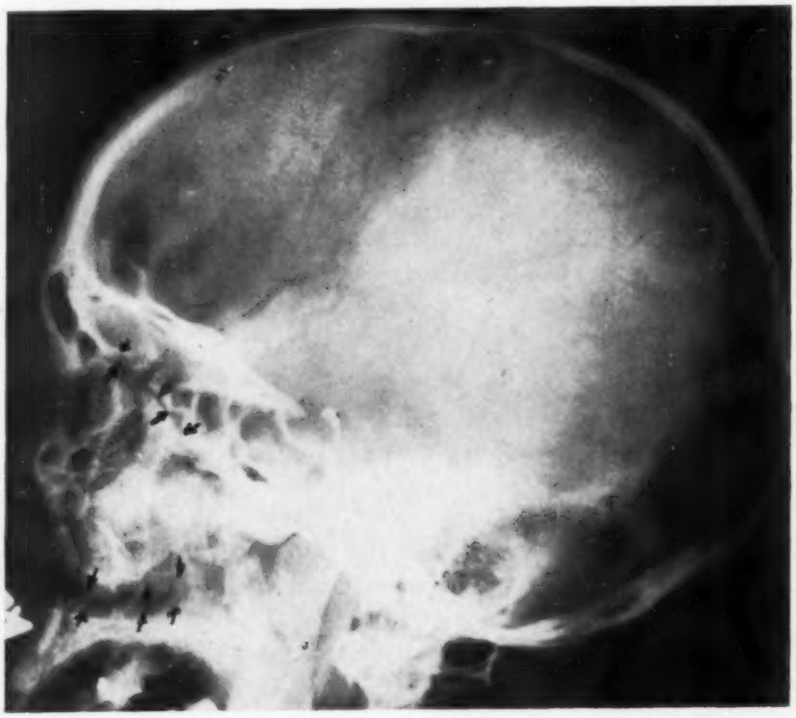

Fig. 138.—Lateral skull x-ray showing fracture separating facial skeleton from the base of skull.

On November 16, while the patient was being given an intravenous infusion of 1500 c.c. 5 per cent dextrose in water, her fractured mandible was reduced by skeletal fixation with local anesthesia and premedication. Monocaine epinephrine was used to anesthetize the mandible by bilateral pterygomandibular blocks, supplemented by subcutaneous injections at the inferior border of the mandible on both sides. The pins were placed very far apart, because the fracture was very oblique. The first two were inserted in the region anterior to the mental foramen on the left, and the second pair in the middle of the horizontal ramus. The distance from the inferior border of the mandible on both sides was about $\frac{1}{2}$ cm. After the pins were inserted, the broken teeth in both upper and lower jaws were removed. No great pains were taken to reduce the fracture by manipulation; the rest of the Frac-Sure apparatus was applied and fastened, to immobilize it temporarily (Fig. 141). After this, the lacerated wound in the skin was revised, and the rubber dam drain removed, since there was no more discharge of pus. The cutaneous sutures in the lip were removed, and the mucosa of the mouth and lip, which was lacerated since the fracture was compounded into the oral cavity, was closed by sutures.

Following this operative procedure, the patient was given a transfusion of 500 c.c. of whole blood. Sulfadiazine therapy was continued.

Postreduction films showed the mandibular fragments in satisfactory position, but there was distraction so that the edges were not approximated (Fig. 140). This was expected and was to be corrected later when reducing the other fractures.

On November 18 the sulfadiazine level in the blood was 5.5 mg. per cent, and it was felt that the patient now (nine days after the accident) was well enough so that we might proceed with the treatment. On November 19, reduction of the fractures of the facial bones was carried out. Preoperative medication consisted of $1\frac{1}{2}$ grains of nembutal two hours before operation, $\frac{1}{12}$ grain of morphine and $\frac{1}{300}$ grain of scopolamine one and one-half hours before operation, and $\frac{1}{300}$ grain of scopolamine and $\frac{1}{120}$ grain of atropine on call. Pentothal sodium intravenous anesthesia was induced. The endotracheal tube was dispensed with because of the nasal injuries. During the operation, however, a nasopharyngeal tube was inserted into the right nostril to facilitate breathing. An incision was made at the external border of the orbit over the frontomalar suture. The malar process of the frontal bone was located, and a small drill hole was made through it, and another in the frontal process of the malar bone where the fracture had occurred. A steel wire was passed through, and the ends wound together after the fracture had been properly reduced. Another drill hole was then made through the external wall of the orbit, and stainless steel wire placed around the margin of the orbit to hold it together (Fig. 144).

The fractured bones in the maxillary sinuses were next reduced. A U-shaped incision was made on the outer wall of the maxilla under the lip; the comminuted anterior wall of the antrum came into view. A piece was removed, and after removing the hemorrhagic material that had accumulated, the remaining parts of the bony wall were pushed back into position by means of

Fig. 139.

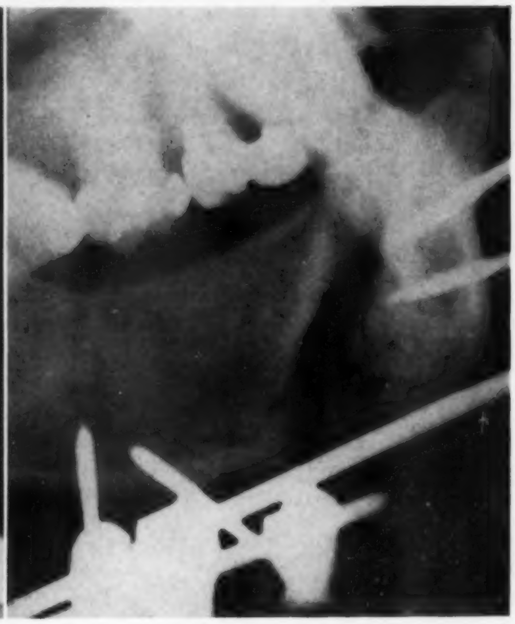

Fig. 140.

Fig. 139.—Fracture of mandible.
Fig. 140.—Fracture of mandible unreduced but immobilized by skeletal fixation.

Fig. 141.—Skeletal fixation applied for temporary immobilization of mandibular fracture.

Fig. 142.—Posteroanterior x-ray showing comminuted fracture of left malar bone and orbit.

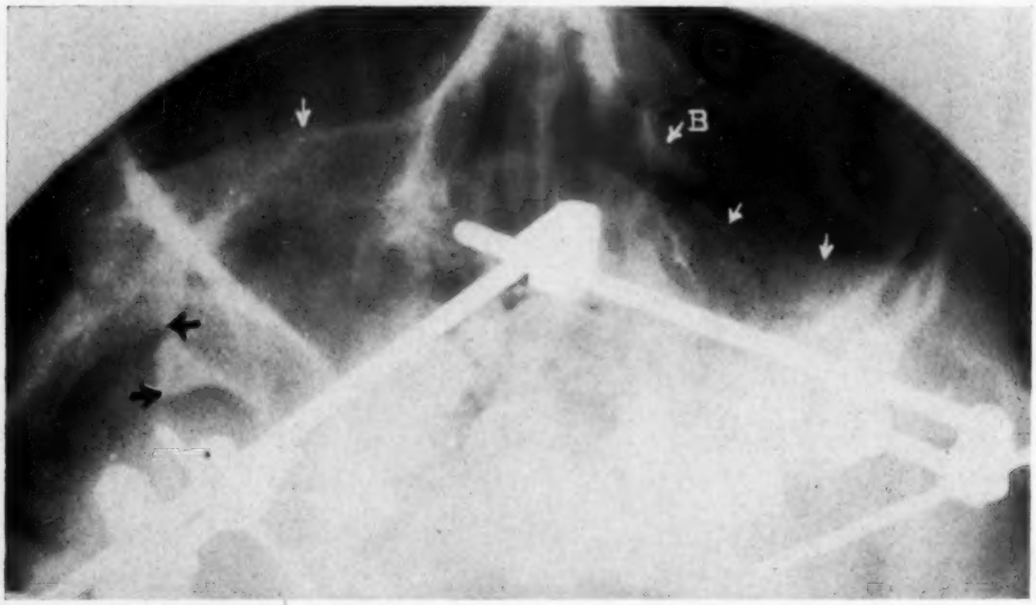

Fig. 143.—Exaggerated Water's position x-ray showing comminuted infraorbital margin on left (white arrows), and displacement of middle face skeleton backward (black arrows).

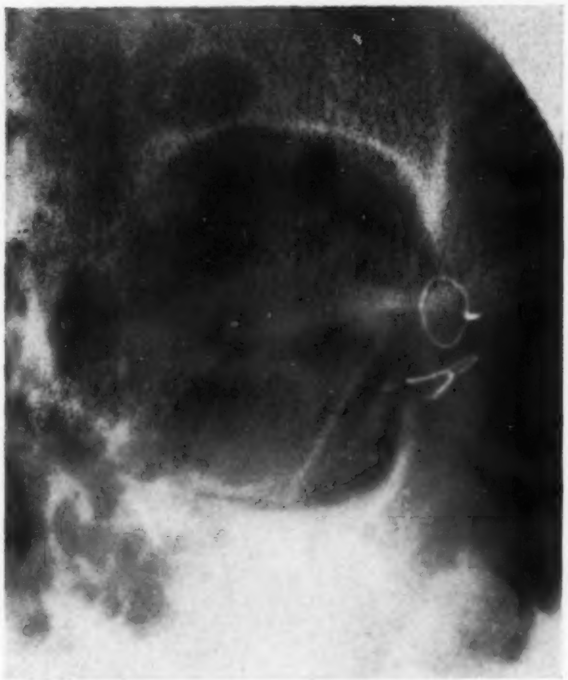

Fig. 144.—Stainless steel sutures inserted to repair orbit.

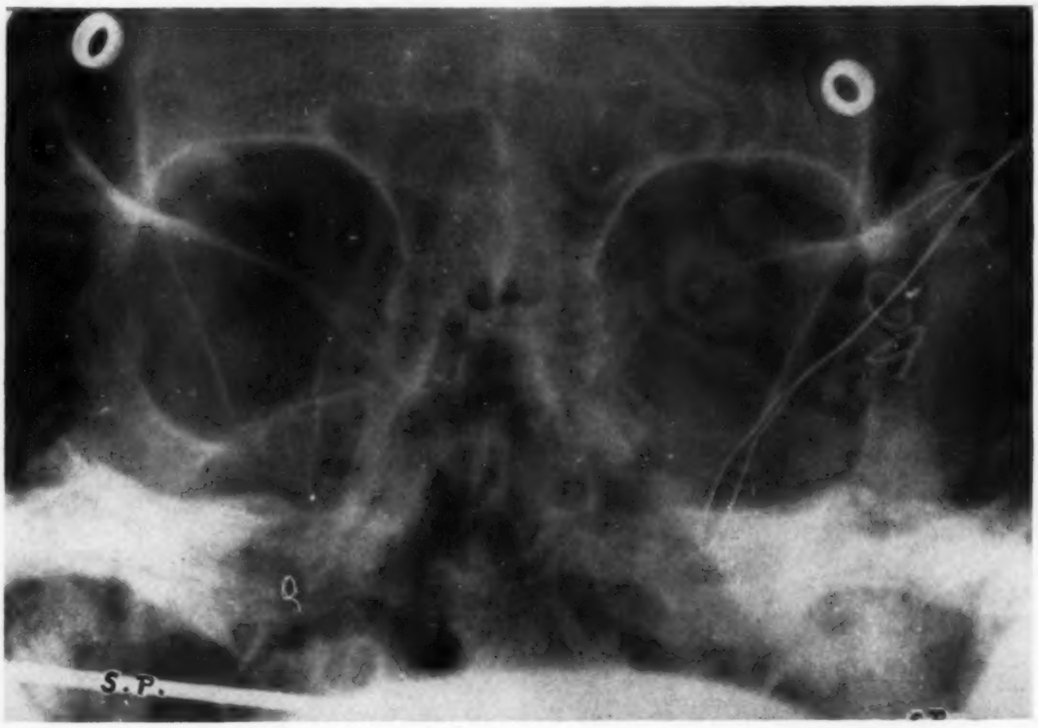

Fig. 145.—Posteroanterior view showing repaired orbit. Looped wire, when attached, will pull up infraorbital margin. *S.P.*, Steinmann pins inserted into alveolar part of maxilla.

finger pressure. The infraorbital ridge, badly comminuted and displaced, was located (Fig. 143) and brought forward; then a stainless steel wire was looped around it and both ends were passed through the skin to hold it in place. The wire was attached to a bar extended from a headband. Fig. 145 shows an x-ray before the wire was fastened to the headband. A lateral view showed the malar bone and orbit in good position.

Next, the mandibular fracture was revised. The Frac-Sure appliance was disconnected, and a circumferential wire was inserted by means of a hypodermic needle, in order to bring the two oblique fragments into contact (Fig. 146). After this, the Frac-Sure appliance was again fastened in the newly reduced position. The antrum was packed with a boric strip to hold the bony fragments in position. The dressing was allowed to extend through the incision.

Fig. 146.—Mandibular fracture completely reduced by means of circumferential wire.

The nasal cavity was carefully examined next. On the left side there were some blood clots, and these were aspirated by suction. At the same time, a piece of bone that was loose was removed. It seemed to be part of the nasal antral wall, probably the corner of the orbital ridge, marked *B* in Fig. 143. An elevator was introduced into the nose to reduce the displacement of the nasal bones. A cotton roll pressure bandage was applied to each side, and fastened down with adhesive tape.

In order to fix the transverse fracture of the maxilla, a Steinmann pin was introduced through the cheek into the freely movable maxillary fragment. Since this could be drilled only halfway, a second one was introduced from the other side. The pins were cut off so as to project about one inch from the cheek. The maxillary fragment could be completely controlled by means of

these wires, which were attached on each side to two rods extended from a Woodard headband, by means of Frac-Sure links (Fig. 147).

Following the operation, the patient received another transfusion of 500 c.c. of whole blood and an intravenous infusion of 1500 c.c. of 5 per cent dextrose and water with $2\frac{1}{2}$ Gm. sodium sulfadiazine added.

Fig. 147.—All fractures reduced: the mandibular by skeletal fixation, the horizontal maxillary fracture by two Steinmann pins inserted through the cheek, and attached to rods extended from the Woodard headband, to which is also attached the wire holding the infraorbital margin.

Postoperative x-rays, taken on November 19, revealed satisfactory reduction. The swelling seemed to have subsided somewhat, and the inward depression of the maxilla seemed to have been completely corrected. On November 23 the temperature remained around 100° F. The patient was still receiving 6 Gm. sulfadiazine daily.

On November 24, the "spiking temperature" still persisted. It was due, no doubt, to a submaxillary abscess located between the line of fracture, which caused a firm, red, swollen area in the submandibular region about one-half the size of the palm. Drainage occurred around one of the pins to which the area extended, and through the small incision in the submental area where previously the rubber dam drain had been inserted. There was no intraoral drainage, and the pins were secure.

The fixation pin in the left maxilla was not secure in the bone, because there was a fracture of the alveolar process in this region, also there was some seepage around the pin externally. This pin, therefore, was removed as it was no longer contributing to fixation, the other pin holding the maxilla satisfactorily in apposition.

The wound at the outer border of the right orbit appeared to be doing well. The patient had some return of sight to the right eye. The ecchymosis and swelling were greatly reduced. The dressings were all changed, and the sulfadiazine and local boric acid treatments to the eye were continued.

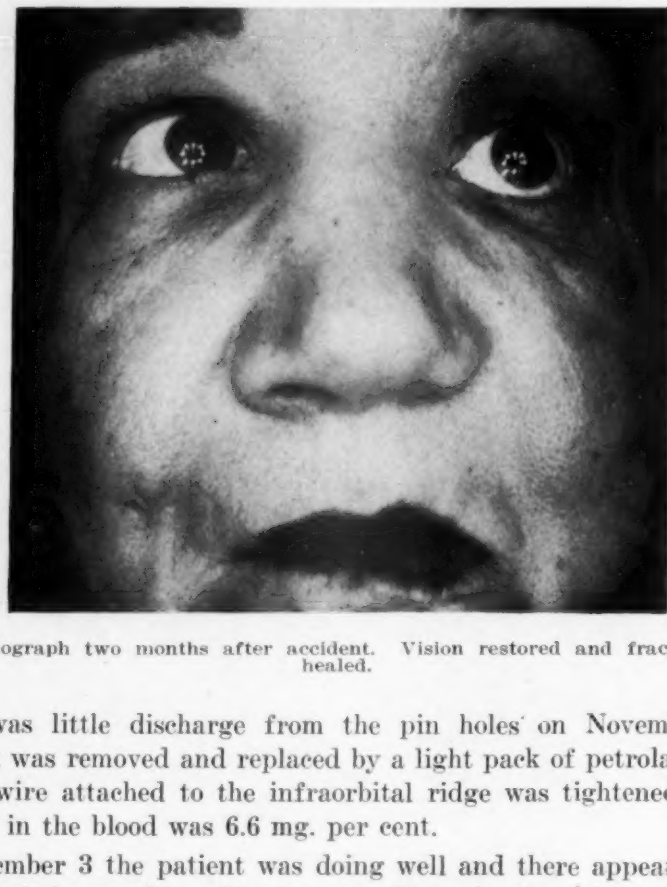

Fig. 148.—Photograph two months after accident. Vision restored and fractures completely healed.

There was little discharge from the pin holes on November 27. The antrum pack was removed and replaced by a light pack of petrolatum iodoform strip. The wire attached to the infraorbital ridge was tightened. The sulfadiazine level in the blood was 6.6 mg. per cent.

On December 3 the patient was doing well and there appeared to be adequate union of the maxilla. The intermaxillary fixation pin on the right, the infraorbital wire, and, therefore, the headcap were removed. The pack in the antrum was no longer needed and was removed also. Skeletal fixation was retained in the mandible.

On December 4 the sulfadiazine was reduced to 4 Gm. per day. The temperature chart was flat. X-rays of the right jaw on December 8 showed the fragments of the mandible in excellent position. Sulfadiazine therapy was omitted.

On December 11 the patient was discharged to be followed in the Outpatient Department. The patient was seen there on December 16. She was getting along exceptionally well. There was considerable callus formation about the

mandibular fracture. The other wounds were well closed, and there was no sign of infection. The mandibular appliance was removed, and the pins were found to be firm. The pin holes were irrigated with zephiran, and dressings were applied.

On December 18 the patient came to the Emergency Ward with a swelling of the floor of the mouth on the right lateral side of the tongue. There was a small fluctuating mass on the right, extending into the floor of the mouth, suggestive of abscess formation. The abscess was incised, and a small amount of pus evacuated, after which a rubber dam drain was inserted. The patient was told to use hot applications at home. Four days later there was little discharge from the rubber dam drain. On December 24, the rubber dam drain was changed, and a new dressing applied.

X-rays taken of the jaws on Jan. 4, 1944, revealed both the fracture of the right mandible and the fractures of the facial bones to be in good position. There was no evidence of osteomyelitis. There was one triangular sliver of bone, apparently fractured off from the lateral lower wall of the left orbit, slightly displaced medially, and another fragment apparently loose and rotated, projecting into the right antrum from the lateral wall. These conditions, however, were asymptomatic and did not require treatment.

Fig. 149.—Drill guard for inserting Steinmann pins and half pins for skeletal fixation.

The patient was told on Jan. 17, 1944, that she should wait about two months before having her upper teeth removed prior to having a denture made. She had entirely recovered from her severe injury. Her vision was tested by the ophthalmological department and found to be normal. She had no paralysis in any part of the face (Fig. 148). The lacerations had left some scars, but the incisions and pin punctures of the skin had healed well, except for a slight stellate scar on the right cheek made by the Steinmann pin.

Discussion.—This patient presents a very severe injury. According to statistics compiled by Blakeslee, based on 595 cases, the death rate in fractures involving the base of the skull with bilateral fixed dilated pupils is 95 per cent; with fixed dilated pupil on one side only, it is 50 per cent. The use of chemotherapy and the very excellent supportive treatment no doubt may have contributed to the favorable outcome in this case. The blindness in the eye and paralysis of the sixth nerve were caused, no doubt, by impingement because of the backward displacement of the middle part of the facial skeleton; reduction therefore relieved the condition. The stellate scar on the right cheek was caused by the insertion of the Steinmann pin, which caught on the tissue. This can be avoided by the use of a guard, such as is shown in Fig. 149. The guard is inserted through the stab incision; its points are engaged in the surface of

the bone, after which the Steinmann pin is drilled into the jaw. The same instrument may be used to insert half pins for skeletal fixation. The hole is drilled by inserting the drill into the tube, and the half pin is similarly placed through the tube. This also eliminates losing the location of the hole, a frequent difficulty.

Case 32

Fracture at Neck of Condyle and Fracture of Mandible Complicated by Osteomyelitis

C. O. (No. 411307), a 44-year-old painter, was admitted to the Massachusetts General Hospital on July 7, 1943, with a chief complaint of pain and swelling of the right lower jaw.

In a fist fight six weeks previously, the patient was struck on the jaw. One tooth was knocked out, and two others became loose and fell out shortly afterward. The skin over the right lower jaw was bruised but not broken. For one week, he had no great discomfort although chewing was difficult and the jaw was slightly swollen. At the end of a week, however, the bruised skin broke, followed by a discharge of blood and pus through the opening. The patient's dentist had x-ray films taken, which revealed a fracture of the jaw. The patient was referred to this hospital, but deferred the visit for four weeks, during which period there was continual drainage of pus from the wound. He had lost 30 pounds since receiving the injury.

Physical examination showed a well-developed and well-nourished man in mild distress. There was swelling and fistula over the right mandibular area (Fig. 150). The right premolar and canine teeth were missing. The remaining teeth were carious and the mouth hygiene was poor. Foul-smelling seropurulent material was draining into the mouth from an open fistula. The heart was found to be enlarged to percussion and there was a blowing systolic murmur heard, loudest at the apex, but transmitted over the precordium and to the axilla. The blood pressure was 125/94. Temperature, pulse, and respirations were normal.

Culture of the discharge showed the presence of nonhemolytic streptococci. the red cell count was 4,000,000. The hemoglobin was 70 per cent. The white cell count was 5,700, with 52 per cent polymorphonuclear leucocytes, 42 per cent large and small lymphocytes, and 6 per cent monocytes. Urinalysis revealed occasional red and white cells. X-rays were taken which showed a fracture of the left ascending rami of the mandible, extending from the mandibular notch to the posterior surface just above the insertion of the stylomandibular ligament (Fig. 152). Another fracture of the horizontal ramus on the right side showed evidence of osteomyelitis in the fracture line (Fig. 151). The left lower molars and premolars were carious and there were many abscesses present.

Sulfadiazine therapy was instituted. Two grams were given on entry, and 1 Gm. every four hours thereafter. In preparation for operation the patient was given $1\frac{1}{2}$ grains of nembutal at bedtime the night before, and $\frac{1}{6}$ grain of morphine sulfate and $\frac{1}{100}$ grain of atropine on call.

On July 8, 1943, under intravenous pentothal sodium anesthesia, two pins were inserted from the outer surface of the face on the left side of the horizontal

Fig. 150.—Contusion and fistula at site of fracture complicated by osteomyelitis.

Fig. 151. Fig. 152.

Fig. 151.—X-ray of mandibular fracture complicated with osteomyelitis.
Fig. 152.—X-ray showing subcondylar fracture of left ramus.

ramus of the mandible about 2½ inches from the fracture line. Two other pins were inserted on the right side of the horizontal ramus of the mandible about 1½ inches away from the fracture line. These two pairs of pins, which were to be used to reduce the fracture in the anterior part of the mandible, were placed far apart because of the osteomyelitis and soft tissue swelling. In order to get intermaxillary fixation for stabilization of the fracture in the left subcondylar region, a pin was inserted into the malar process of the left cheek. After the pins were inserted, a number of teeth were extracted; namely, an upper molar which was decayed and abscessed, and, in the mandible, four roots and three teeth were either abscessed or loose, or extended into the fracture line. It was because of the loss of these teeth that it was impossible to use intermaxillary fixation. A sequestrum was then removed in the anterior part of the mandible from an oral approach. The fistula in the chin was then excised, and two more very long narrow sequestra were removed from the outside. After débridement of the fractured area, sulfanilamide and sulfathiazole powder were inserted between the fracture line. A rubber dam drain was placed and sutured to the skin with two silk sutures.

The connecting links of the Frac-Sure appliance were then applied, to reduce the fracture in the anterior part of the mandible, after which a rod was attached to this appliance, connecting it with the pin inserted into the malar bone (Fig. 153). Since it was feared that this unilateral fixation was not quite adequate, a Barton bandage was applied.

The second day after the operation, the temperature rose to 104° F., the pulse rate to 116, and the respiratory rate to 30. There was thought to be some clinical evidence of atelectasis. X-ray examination of the chest was negative, but in spite of this, suction was instituted and a considerable amount of bloody sputum was aspirated. The following day the temperature dropped to 100° F., the pulse rate to 84, and the respiratory rate to 24. The postoperative x-ray is shown in Fig. 154. Sulfadiazine therapy was continued until July 14, when the urine contained numerous red blood cells. The sulfadiazine level in the blood at this time had reached 9.9 mg. per cent.

On July 14 the immobilization of the mandible was definitely found to be inadequate. Too much strain was placed on the one pin in the malar bone, and the fixation of the mandible with the pins so far apart was not as strong as was necessary in order to get union in the anterior part of the mandible. Therefore it was decided that a second pin should be inserted on the right side into the malar bone in a similar way to that inserted on the left.

The pin was inserted under intravenous pentothal sodium anesthesia, with preoperative medication again consisting of 1½ grains of nembutal at bedtime the night before, and ⅙ grain of morphine sulfate and $\frac{1}{100}$ grain of atropine on call. Two bars were attached to this pin and the appliance on the mandible, and a second bar was added on the left to form on each side a triangle that would hold the mandible uniformly stabilized (Fig. 155).

On July 19, the rubber dam drain was removed from the incision under the jaw, as drainage had ceased at that time. The patient was discharged on July 23 to be followed in the Outpatient Department. There was slight seepage from around the pins in the mandible.

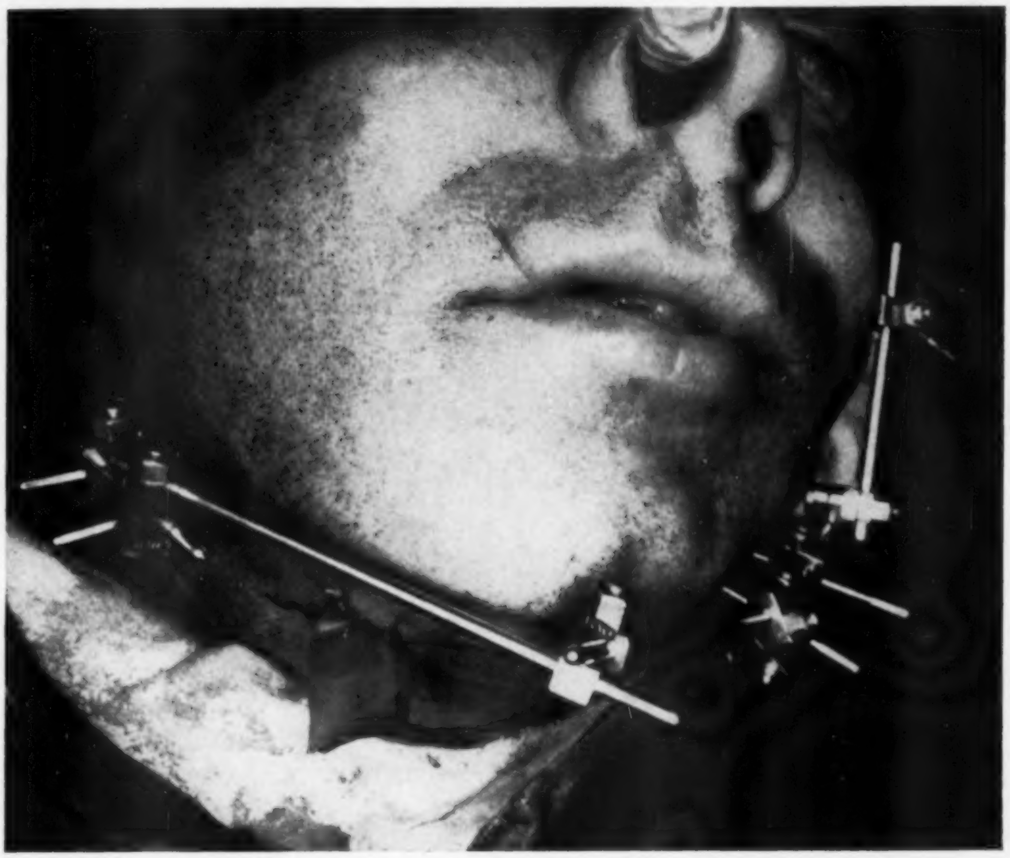

Fig. 153.—Skeletal fixation by means of Frac-Sure appliance following sequestrotomy and reduction. Note incision under chin with rubber dam drain sutured to the skin. A pair of half pins is inserted on each side of the mandible far from the area of osteomyelitis. A bar is attached to a pin inserted into the zygoma to immobilize the jaw.

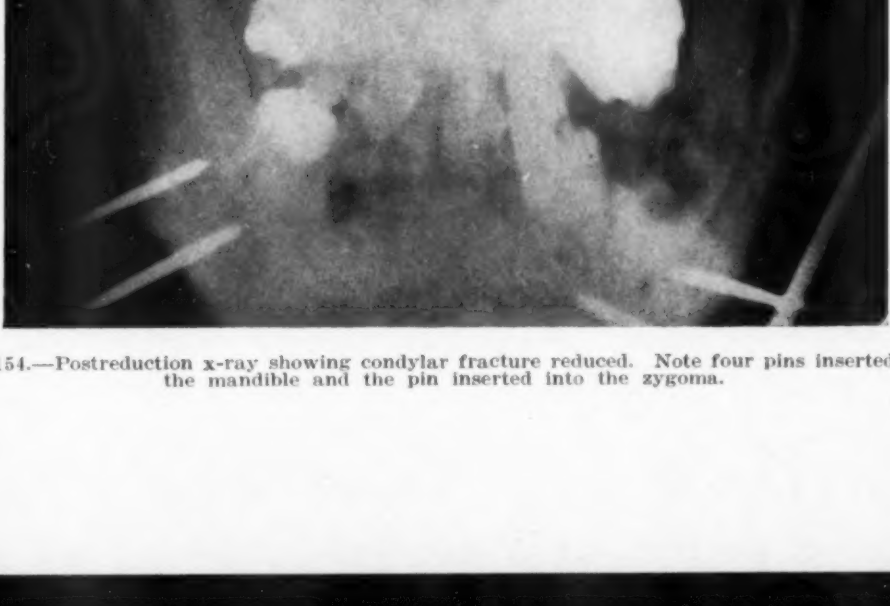

Fig. 154.—Postreduction x-ray showing condylar fracture reduced. Note four pins inserted into the mandible and the pin inserted into the zygoma.

Fig. 155.—Mandible more completely immobilized by means of a pin in each zygoma. On each side two connecting rods extend to a Frac-Sure appliance on the mandible, forming a triangle for strength.

Fig. 156.—Photograph of right cheek, showing absence of dimpling.

The patient was seen on July 26 in the Outpatient Department. Examination showed some union at the fracture site on the right side of the lower jaw. Dressings were changed and the skin wounds around the pins irrigated with hydrogen peroxide.

Two sequestra of alveolar bone were removed from the midline of the mandible on August 6. X-rays taken the same day showed that there was considerable bony resorption present where the metal pins had entered the jaw. The fracture of the condyle had healed.

On August 12 the zygomatic pins were removed, along with the four bars by which they were connected with the mandible. A week later, the skin over the malar bone where the pins had been inserted had healed well, although the skin was attached to the bone and formed a very small dimple on the left. The fracture in the mandible was examined, and it was found that there was fairly good fibrous union. Because of the loss of bone due to osteomyelitis, approximation was prevented, and it was felt that the patient would probably not get bony union without a bone graft being inserted. However, after the appliance had been removed from the mandible the jaw felt quite comfortable and firm. One pin on the right near the infected area was found to be loose, while the pins on the left were solid. The skin perforations and the holes in the bone were treated with zephiran.

X-rays taken on the same day revealed the fractures to be held in good position. A space of about ½ cm., not as yet filled with bone, was seen. On the right side of the horizontal ramus the fragments appeared to be separated.

By October 28 the patient had perfect union of both fragments, but there was still a small amount of discharge from the infected pin wound, which was treated with zephiran until it healed.

On December 8, in order to prepare the patient for dentures, the lower left canine and lateral incisor were removed under local anesthesia. The right lower second molar was extracted on December 16, and the lingual crest of the lower right mandible in the molar region was trimmed on December 23. When the patient was seen on Jan. 20, 1944, his condition seemed to be good.

Discussion.—The use of skeletal fixation in this patient was indicated because he had an insufficient number of good teeth for intraoral intermaxillary fixation. The result must be considered excellent in view of the fact that the fracture was six weeks old and complicated by extensive osteomyelitis and submandibular infection.

Skeletal fixation in this patient was used also for immobilizing the mandible, which was necessary not only because of the condylar fracture, but also to give better fixation for the mandibular fracture.

Dimpling is generally due to pin seepage. In this patient it occurred on the left cheek, because the pin was under too great a strain here. On the right no scar can be seen today (Fig. 156). In the mandible, dimpling occurred because of the subcutaneous swelling which is always associated with a septic fracture. It was not bad, however, and, if necessary, can be remedied.

II. CASES OF OSTEOMYELITIS

Case No. 33

Chronic Osteomyelitis of the Mandible

F. R. (No. 407044), a 58-year-old man, presented himself at the clinic on June 3, 1943, with complaints of pain and swelling of the jaw, which were of two months' duration and had started with a toothache.

About three months before admission, he broke a fixed bridge made to restore the lower incisors. The root of the abutment tooth on the left, to which the bridge was attached, came out with the bridge. The soreness which he had experienced for about six months was relieved when the bridge was removed.

Two months before admission, the site where the tooth came out began to feel sore. The extraction wound had never healed, and little mounds of "gum tissue" rose in the socket. The patient's dentist packed this with sulfanilamide dressing, after breaking the granulations. Pus began to discharge, however, and continued to do so until the time of admission. He had used hot saline mouth washes without any improvement.

About six weeks before, the patient was put to bed and given sulfadiazine for a week. Meanwhile, he noticed a swelling underneath his jaw. Flaxseed poultices were applied to this area, and small gingival abscesses continued to form, which would break and discharge a seropurulent material.

On examination, his breath was found to be foul. He stated that the pain in the jaw as well as the swelling on the left side of his face was causing considerable discomfort, so that he required frequent sedative medication. In spite of this, he had been sleeping poorly. His appetite had been good, but eating was very painful. In the past few months he had lost about 20 pounds.

He had not had chills or fever except on one occasion, five days before admission, following the injection of a diuretic by his local physician.

Diuresis was profuse but was accompanied by a severe chill followed by a temperature of 102° F.

The past history of the patient revealed that he had always been susceptible to infections, and cuts on his hands took some time to heal.

Examination of the mouth revealed that the maxilla was edentulous. In the mandible the roots of four lower teeth remained on the right. These were quite carious. The mucous membrane on the left side of the lower jaw was swollen and inflamed. In the region of the left canine, there was a sinus tract from which creamy pus could be expressed. There also were other small places on the left which were discharging pus. A firm, fluctuating, nontender, swollen area, measuring about 3 cm. in diameter, was seen in the submental region.

Other significant factors concerning this patient were as follows: He had been in bed for the past six weeks chiefly because of cardiac disease which came on after an attack of bronchitis. He was found to have hypertension and had not yet reached a state of compensation.

An x-ray of the jaws, taken on June 5, 1943, showed rarefaction of the bone about the root remnants in the right lower jaw. There was no evidence of de-

struction of the bone by osteomyelitis. The stylohyoid ligaments showed evidence of calcification.

On June 8, 1943, the lower remaining roots that could be identified were removed, using novocain block anesthesia. Two days later, by pressing on the submental induration, pus could be expressed readily from the tooth sockets.

Another x-ray taken on June 11, 1943, showed the bone structure in the left anterior aspect of the lower jaw to be somewhat irregular. There was only one area in which there seemed to have been some destruction not seen on the other previous films. However, the projection was somewhat different. The findings were not very definite, but it was felt that they represented early osteomyelitis. Oral sulfadiazine treatment was carried out for the following four days.

On June 13 the patient was admitted to the House. He was given sulfadiazine, starting on June 14 through June 17, 1 Gm. every five hours. On June 15, under local anesthesia, he was operated on for incision and drainage of the submental abscess. Premedication consisted of $\frac{1}{6}$ grain of morphine and $\frac{1}{100}$ grain of atropine. After the usual preparation of the skin, an external incision was made in the median line in the submental area. The abscess was opened by means of a hemostat, and found to extend between the platysma and mylohyoid muscles. Pus was evacuated from the incision, and cultures were taken for aerobic and anaerobic growth. A rubber dam drain was inserted and sutured to the skin. An intraoral incision was made in the left anterior region, and a rubber dam drain inserted here also; on the right a sequestrum was removed, after which sulfanilamide powder was placed in the wound, followed by a boric strip.

The bacteriologic findings on the culture made from the submental abscess showed it to be due to nonhemolytic streptococcus. Chemotherapy was continued and supportive treatment was administered, consisting of irrigations with Dobell's solution, alternating with saline solution.

On June 21 the patient was discharged, to be followed in the Outpatient Department. Here, on June 30, the rubber dam drain in the incision under the chin was changed. It was still in position on July 2, but drainage was subsiding. The patient said that two small pieces of bone had worked out of the fistula within the mouth.

X-rays at this time showed extensive areas of bone destruction in the anterior and medial part of the lower jaw. There were several areas of increased density within this area of bone destruction, which had the appearance of sequestra. There was a line of demarcation ascending somewhat posteriorly, suggesting that a large part of the jaw anterior to this bone might sequestrate. The jaw posteriorly showed areas of increased density consistent with longstanding infection. The findings were those of osteomyelitis.

On August 31, there seemed to be some movement of the infected bone. In September a large piece of necrotic bone became exposed, projecting through the oral mucosa in a bizarre fashion (Fig. 157).

X-rays, taken on October 6, showed that the entire anterior portion of the jaw formed a sequestrum which apparently had separated from the rami. It

Fig. 157.—Photograph showing partly sequestrated bone in case of chronic osteomyelitis of mandible.

Fig. 158.

Fig. 159.

Figs. 158 and 159.—X-rays showing osteomyelitis with large sequestrum.

Fig. 160.—Wound, from which sequestrum has been removed, is healing.

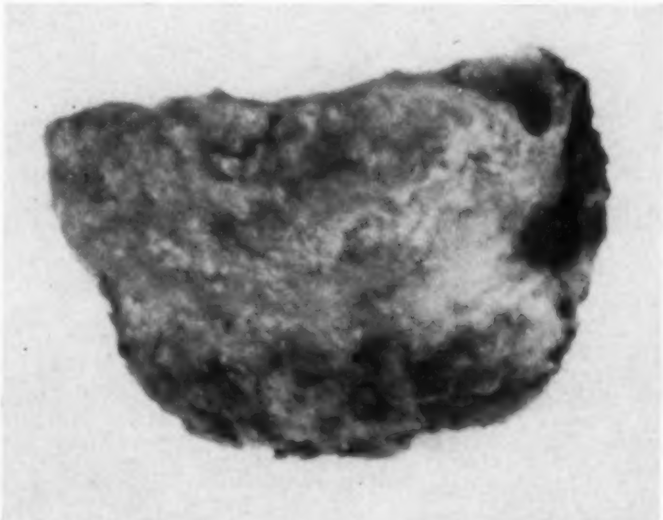

Fig. 161.—Photograph of bone sequestrum.

was quite dense, and was probably about to exfoliate. There was a bony involucrum about part of it (Figs. 158 and 159).

On October 25, the sequestrum was removed with little hemorrhage and without causing any fracture. One week later, the bed from which the sequestrum had been removed was well closed, and there were no signs of suppuration, although the patient complained of some pain. The necrotic end of the jawbone on the left was still exposed (Fig. 160); on the right it had healed over.

Pathologic Examination.—The specimen consisted of a piece of bone which showed evidence of erosion. One large unhealed tooth socket was seen at one end, and two others partly healed at the other. It had the appearance of a sequestrum (Fig. 161).

Bacteriologic culture of the diseased bone contained abundant alpha hemolytic streptococci, few beta hemolytic streptococci, and *Staphylococcus aureus*

The course of the disease from here on was uneventful, and asymptomatic. A spicule of bone on the right was found to be mobile on November 17; it was removed. At present there is good evidence of new bone formation, and the prognosis may be considered very favorable, with possible sequestration later of one or two small spicules of diseased bone.

Discussion.—The treatment in this case was long drawn out. Much patience was needed, and restraint in not performing a surgical operation too soon. Nothing but a resection of the anterior part of the mandible would have arrested the process. Nature, in its own way, gradually detached the necrotic bone, and formed an involucrum which held the bone together after the sequestrum had separated and was ready to be expelled. Surgical interference might well have resulted in fracture if performed too early, while at the correct time the sequestrum could be lifted out without the use of force and without destroying the pyogenic membrane that had been built up around it to protect the adjoining normal bone.

III. CASES OF NEURORRHAPHY

Case 34

Anesthesia and Paresthesia of Lip Following Malunited Fracture of Mandible, Treated by Neurorrhaphy

G. K. (No. 424105), a 60-year-old man, reported to the Outpatient Department on Oct. 21, 1943, complaining of intermittent burning pain and numbness of the lower lip and alveolar ridge of the mandible.

Two years ago the patient was slugged and robbed. His face swelled considerably, and two days later a bilateral fracture of the jaw was diagnosed at a local hospital. This fracture had been treated by a head bandage and apparently had healed satisfactorily. One year ago, the patient said that he had had an automobile accident in which his leg and jaw were injured. He had sustained a simple mandibular fracture on each side in the canine region of the edentulous jaw, approximately at the site of the previous fracture. These frac-

tures healed without treatment, but the patient, about six months before admission, began to have tenderness over the fracture site, especially on the right, and to a lesser degree on the left. A light touch caused a burning sensation both on the outside and the inside of the right lower lip. He was unable to bite hard food because of pain.

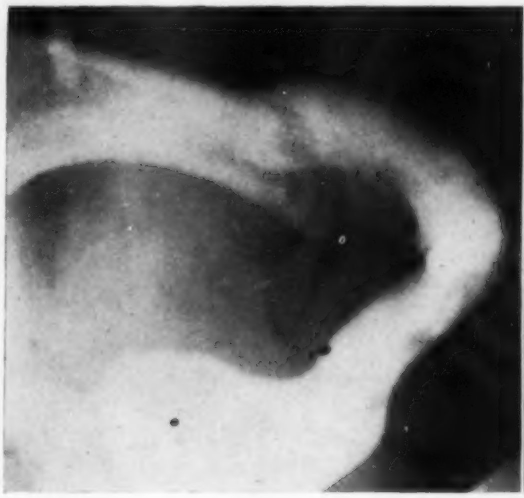

Fig. 162.

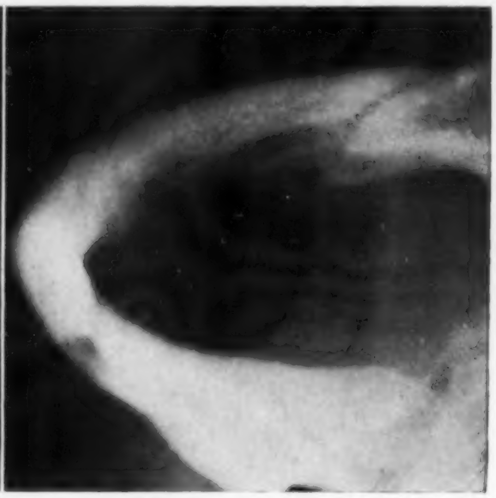

Fig. 163.

Fig. 162.—X-ray of right mandible showing malunion and disarrangement of mandibular canal causing neurological complaints.

Fig. 163.—X-ray of left mandible showing malunion and large resorptive area enlarging mandibular canal.

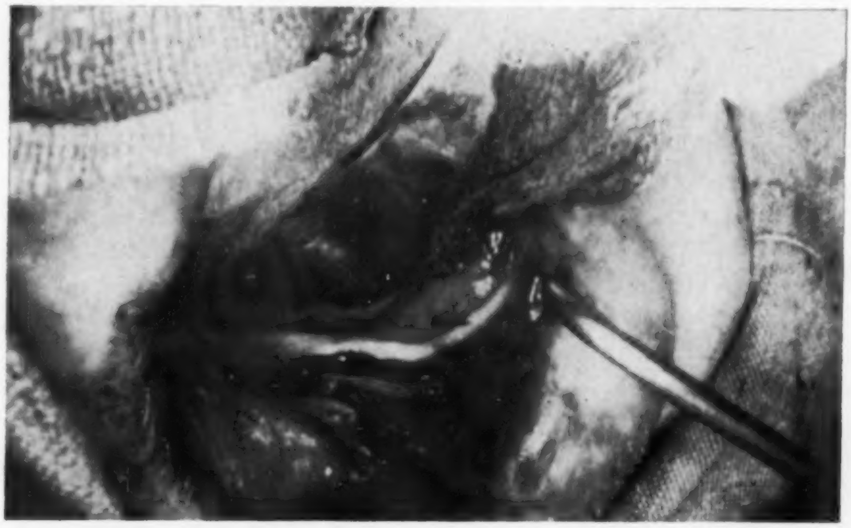

Fig. 164.—Photograph showing the long window cut in the side of the mandible, exposing the posterior part of the mandibular nerve.

The examination revealed a slender ramus of the mandible, due to alveolar atrophy, which had caused a decrease in vertical dimension. There was no crepitus and the union seemed good, although there was an irregular projection at the alveolar crest.

The x-ray report of Oct. 21, 1943, said that the old fractures on either side of the mandible showed satisfactory union. The x-rays were reviewed, however, to study the cause of the neurological signs, and it seemed that malunion on the right side (Fig. 162) near the angle of the jaw had prevented the mandibular nerve from uniting, while on the left (Fig. 163), where a large resorptive area had formed, the nerve probably had united. An operation to repair the right mandibular canal and join the severed ends of the mandibular nerve was advised.

The patient was admitted to the House on Nov. 8, 1943. On November 9 he was operated upon under intravenous pentothal sodium anesthesia. The preoperative medication was 1½ grains of nembutal two hours before operation, $\frac{1}{300}$ grain of scopolamine one hour before operation, and $\frac{1}{300}$ grain of scopolamine and ⅛ grain of morphine sulfate on call. His mouth was perfectly dry, due to the scopolamine and atropine. The oral mucosa was carefully prepared with iodine solution. An incision was made along the alveolar border on the right side of the mandible from the ramus to the canine region. With a drill, holes were made in the side of the mandible to outline a long window, which was completely cut out by means of an osteotome. The mandibular nerve was then visible in the posterior end of the mandible (Fig. 164) and could be lifted out of the canal except where it was attached in the scar tissue between the fracture line. Next, a window was cut in the anterior part of the mandible, but when the nerve was lifted out of the canal, it broke at the place where the mental nerve is given off. Thus, the anterior part of the nerve, severed from its alveolar branch, was seen to extend to the mental foramen. Both the severed ends were carefully dissected out of the scar tissue. The canal was repaired through the part where the fracture had disturbed its continuity, and, when both ends of the nerve were placed into proper position, they were found to approximate perfectly. The mucoperiosteum finally was replaced, and sutured with a continuous silk suture.

The day after the operation, the patient said that he had less pain in the right mental region, and his jaw felt better. There was little postoperative swelling and the patient seemed to do well. He was discharged on the third postoperative day to be followed in the Outpatient Department.

He was next seen on Nov. 17, 1943. At that time he said that he was greatly relieved, his paresthesia having been relieved, and there being only anesthesia. The sutures were removed and the patient told to return in about two months. On December 10, the patient reported that he felt pin and needle sensations, indicating a return of sensation.

Discussion.—The prompt relief of the paresthesia was probably due to the complete severing of the nerve where it was impinged in the scar tissue at the site where the fracture had occurred. Union, however, had not as yet occurred; therefore, the anesthesia of the lip persisted. It is to be expected that if union takes place, the numbness will disappear. This is the first attempt made by us to repair the mandibular nerve in an old malunited fracture with neurological complaints.

Case 35

Anesthesia and Paresthesia Persisting for Eighteen Months, Treated by Neurorrhaphy

S. H. (427252), a 55-year-old woman, came to the Outpatient Department on Nov. 5, 1943, complaining of numbness of the lower lip on the left side.

Some eighteen months before admission she was in an automobile accident, in which her left ninth rib, right wrist, and left mandible were fractured. The fractured mandible was treated by intraoral wiring. Following the mandibular fracture, the patient's lip was quite sore and numb. Both the soreness and numbness had remained. She had periods when she felt drawing sensations, and often she felt as if the jaw was swollen.

X-rays were ordered and the following report was received: "A metal wire is present in the left lower mandible, there being some bony resorption about it. The fracture line has almost disappeared." Further study of the x-ray, which is reproduced in Fig. 165, showed the mandibular canal emerging on the surface of the alveolar ridge just where the wire is twisted together. From this finding it was surmised that the nerve which had been severed by the accident had not united.

Removal of the wire and neurorrhaphy were advised, and the patient was admitted to the House the day before operation. The preoperative medication consisted of $\frac{1}{8}$ grain of morphine and $\frac{1}{100}$ grain of atropine on call. On Nov. 30, 1943, a neurorrhaphy was performed under gas, oxygen, and ether anesthesia. An incision was made over the alveolar crest about $1\frac{1}{4}$ inches in length, with vertical extensions on the outside of the jaw. The mucoperiosteum was reflected to expose the mandible. A window was cut by means of an osteotome to expose the mandibular nerve on each side of the fracture. The wire which had been used for internal wiring of the fracture was located and removed. The posterior part of the nerve was seen to emerge from the canal and extend down on the lingual surface of the mandible between the periosteum and mucosa, terminating in a small swelling. This was removed by cutting the nerve at the place where it appeared normal. The anterior part of the nerve was then exposed, and dissected free from the fibrous tissue in the fracture line. After the mandibular canal was repaired so as to make it continuous, it was found that the two nerve ends were still of sufficient length to be approximated when placed into the newly formed canal. A suture was attempted with fine tantalum wire, but, since the needle was too coarse, the nerve split; therefore, the two nerve endings were placed in contact without being sutured. Since they were protected by the walls of the mandibular canal on each side, it was felt that they could not be displaced very much. The mucoperiosteum was sutured over the bone with a continuous silk suture.

Pathologic examination of the tissue swelling attached to the cut-off end of the nerve showed this to consist of connective tissue. Therefore, the possibility of its being an amputation neuroma, which had been entertained, was not confirmed by the findings.

The postoperative course of this patient was uneventful. She was discharged on the third postoperative day to be followed in the Outpatient Depart-

ment. An x-ray was taken at this time, showing the wire removed and the mandibular canal repaired (Fig. 166).

On Dec. 6, 1943, the patient reported that she was considerably improved. Her painful sensations had disappeared, but she still had some numbness of the

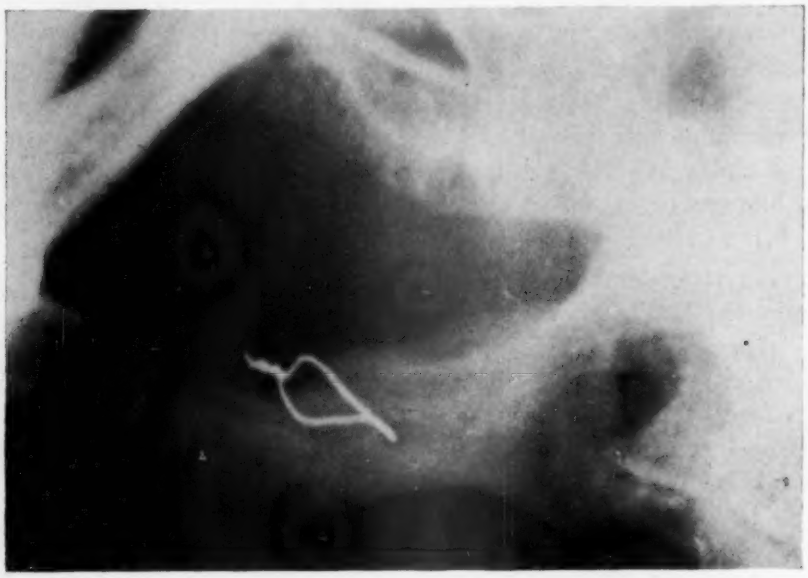

Fig. 165.—X-ray showing intraoral wiring employed for fixation of fracture. The mandibular canal emerges on the surface of the alveolar ridge under the wire.

Fig. 166.—Postoperative x-ray showing wire removed and mandibular canal repaired.

lip, which, of course, was to be expected. On December 23, she reported that not only her paresthesia, but her anesthesia had entirely disappeared. She also stated that she had no more of the drawing sensations and periodic swelling.

Discussion.—This is the second case of neurorrhaphy. Though the anesthesia and paresthesia had been in existence for eighteen months, the patient

had prompt relief. The experience gained by the first operation helped to improve the technique. The short time in which healing occurred is evidence of the wonderful power of regeneration of the trigeminal nerve branches.

IV. REMOVAL OF FOREIGN BODY

Case 36

Removal of Broken Needle From Pterygomandibular Region

M. G. (No. 421790), a 20-year-old girl, came to the Outpatient Clinic on Sept. 29, 1943, stating that a needle had been broken off in the pterygomandibular space on the right.

Two days before admission, during the attempted administration of novocain for a right mandibular block, a needle was broken off by her local dentist. Two attempts had been made to remove it, one by the local surgeon, who operated for two hours without finding it.

Upon examination, the jaw was found to be swollen and tender, and the patient was unable to open her mouth fully. There was some swelling internally over the ramus of the mandible, the mucous membrane was reddened, and there was some yellow exudate. On the following morning the patient's jaw was still swollen, and there was some increase in the muscular trismus. It was feared that there might be some sepsis, which would be followed by abscess formation later. Therefore, hot mouth irrigations were administered, and chemotherapy consisting of an initial dose of 1 Gm. of sulfadiazine every six hours was instituted. The white blood count was 11,411 on admission. X-ray examination showed the needle; apparently it had moved to the posterior limit of the pterygomandibular space (Fig. 167).

The patient was discharged on September 30 because it was generally felt that the inflammatory reaction should be allowed to subside before further exploration. At the time of discharge the patient was advised to return to the Outpatient Department ten days later, for an evaluation of the case and a decision of whether or not to operate.

On October 8 the patient returned to the Outpatient Clinic. There was no appreciable swelling left, though the old wound in the retromolar fossa of the lower right mandible was still reddened and had edematous margins with a seropurulent discharge. There were no palpable or tender lymph nodes. She was advised to receive further treatments of warm saline irrigations and hydrogen peroxide from her local dentist.

The patient was admitted to the House for the second time on October 21, for the removal of the foreign body from her jaw. The next day, the operation was performed under intratracheal nitrous oxide, oxygen, and ether anesthesia. The preoperative medication consisted of 1½ grains of nembutal at bedtime, ⅙ grain of morphine and $\frac{1}{100}$ grain of atropine on call. A Kazanjian indicator (Fig. 168), which had previously been prepared by inserting a pointed metal wire into the tube of an Angle band, was attached to the right lower second

Fig. 167.—X-ray showing broken-off needle far back in the pterygomandibular space.

Fig. 168.—Foreign body location. Kazanjian indicator applied to mandible.

molar. The point was introduced into the pterygomandibular space in the same manner as that used for making a mandibular block injection.

An x-ray film was taken while the patient was on the operating table, first from the lateral (Fig. 169), and then from the anteroposterior aspect (Fig. 170). These showed the indicator crossing the needle approximately in the middle.

Fig. 169.—X-ray taken during the operation (note intratracheal tube) with indicator, *A*, in position; it crosses needle, *B*.

Fig. 170.—Anteroposterior view taken during the operation, showing relation of indicator, *A*, to needle, *B*.

An intraoral incision was then made along the anterior border of the ramus, and by means of blunt scissor dissection, the pterygomandibular space was opened. A three-pronged tracheotomy dilator was inserted and opened (Fig. 171), which allowed dissection under direct vision. The indicator was located, after which it was easy to find the broken-off needle with a curved probe. When it came into view, it was grasped with small Kelly forceps and removed. Sulfanilamide powder was dusted into the wound, and a knotted rubber dam drain was inserted to prevent the formation of a hematoma.

Fig. 171.—Tracheotomy dilator used to remove foreign body under direct vision.

The day after the operation, the patient had little swelling. Earlier, she had experienced some numbness of the tongue, but this had disappeared. The drain was removed twenty-four hours after the operation, and the patient was discharged on the first postoperative day.

Discussion.—We are presenting herewith a method for the removal of a broken-off needle, which can be used for the removal of other foreign bodies. It consists of a method of locating the foreign body by x-rays, taken from two different views during the operation, with an indicator attached that will remain in position if the patient opens the mouth or moves while the film is being developed. After the position is ascertained, the use of the tracheotomy dilator makes it possible to separate and retract the tissues, so that the foreign body can be located by direct vision, instead of relying on the sense of touch for finding and removing it.

V. SKELETAL DISEASES AFFECTING THE JAWS

Case 37

Paget's Disease Involving the Maxilla

A. F. (No. 321546), a 56-year-old widow, was admitted to the House on Nov. 15, 1943, her chief complaint being inability to wear her upper plate because of bony deformity.

In 1926, a swelling on the right side of the patient's face was noted by her physician. As there were no symptoms, the patient did nothing about this until 1931, when, by examination in the Outpatient Department, a lump the size of a cherry was found in the upper maxilla. Laboratory studies at that time showed the blood calcium was 10.5 mg. per cent, the phosphorus was 2.73 mg. per cent, and the phosphatase 18.32 units. X-rays were taken, and interpreted as showing Paget's disease with localization in the maxilla. The patient was studied in the Eye and Ear Infirmary during this time.

Her first Massachusetts General Hospital admission was in March, 1933. She complained of jaw pain of six days' duration. The diagnosis was Paget's disease of the skull and right inferior maxilla, a right maxillary alveolar abscess, and chronic right otitis media. An operation for the extraction of a tooth and the maxillary mass was performed.

The second M. G. H. admission of this patient was on Feb. 16, 1942. This time, she entered because of a tumor beneath her left cheek of five years' standing. X-rays of the skull showed Paget's disease, and x-rays of the jaws revealed periapical abscesses. To the diagnosis made in 1933 was added dental caries, obesity, and hypertension. Three teeth were removed. The blood calcium level was found to be 10.5 mg. per cent, phosphorus 3.1 mg. per cent, and phosphatase 63.5 units. After discharge, the patient was followed in the Outpatient Department. She complained of left upper arm pain. She was given one dram per day of 8 per cent thiocyanate solution. On Oct. 18, 1943, her blood calcium level was 9.4 mg. per cent, phosphorus 2.4 mg. per cent, and phosphatase 107.6 units. The increase in the phosphatase level to 107.6 units was a circumstance which favored the diagnosis of Paget's disease.

After operation in 1942, the growth in the maxilla continued. There was a marked expansion on the outer surface on the left, and a smaller bulge on the right. The patient was unable to wear her upper denture (Fig. 172). She had been able to eat hard and soft foods, being inconvenienced at times, however, by abrasions of the mucosa from chewing hard foods. She had been in good health generally and had worked hard at home, although she was somewhat weak since she had been on a 1,500-calorie M. G. H. reducing diet for the past month.

The third M. G. H. admission of this patient was on Nov. 15, 1943. She entered for an alveoplasty to permit the wearing of a maxillary denture. X-rays taken at this time are shown in Figs. 173 and 174.

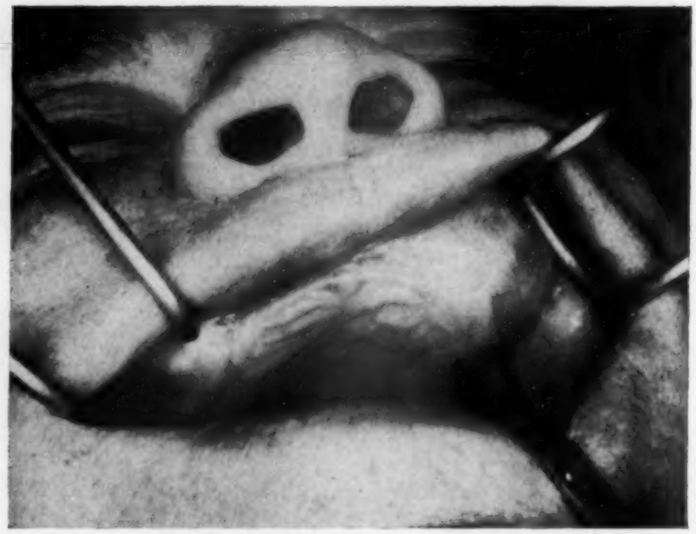

Fig. 172.—Paget's disease causing expansion of maxilla.

Fig. 173.—X-ray showing Paget's disease affecting calvaria and maxilla.

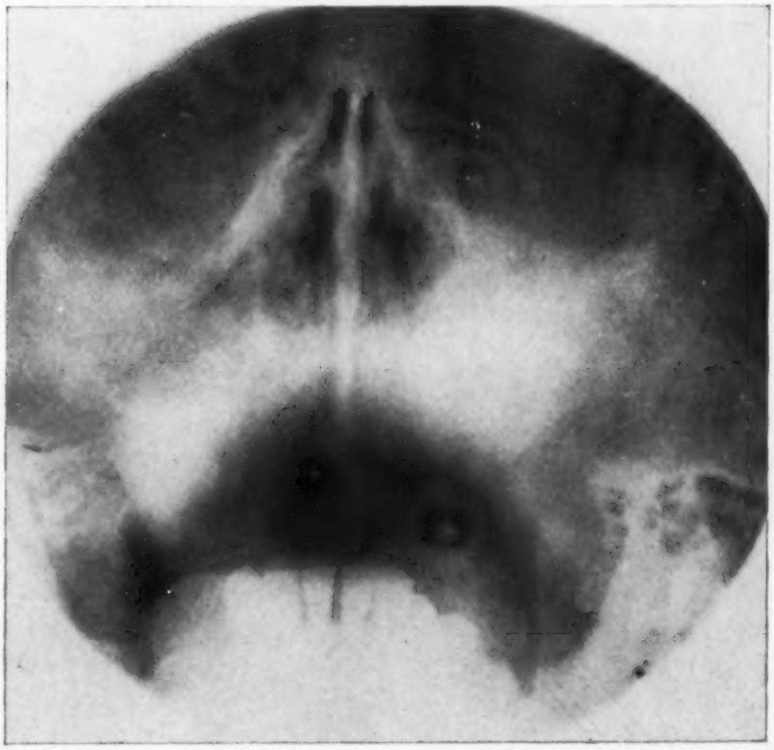

Fig. 174.—X-ray showing Paget's disease involving maxilla and maxillary sinuses.

Fig. 175.—Retraction of the mucoperiosteum showing honeycombed, soft bone.

The patient was operated upon Nov. 16, 1943. The preoperative medication consisted of $1\frac{1}{2}$ grains of nembutal two hours before operation, and $\frac{1}{6}$ grain of morphine sulfate and $\frac{1}{200}$ grain of scopolamine on call. Local anesthesia was used; two infraorbital, two zygomatic, and three palatine injections were given to anesthetize the entire upper jaw. An incision was made along the alveolar border from the left to the right and extended vertically on each side. The mucoperiosteum was detached by means of a periosteal elevator, and the bulging bone became visible (Fig. 175). This was cut down by means of chisels, and was found to be very soft so that it could be easily carved. After the normal contour of the alveolar process and bone was restored, the mucosa was replaced and sutured with silk. The same procedure was repeated on the right side.

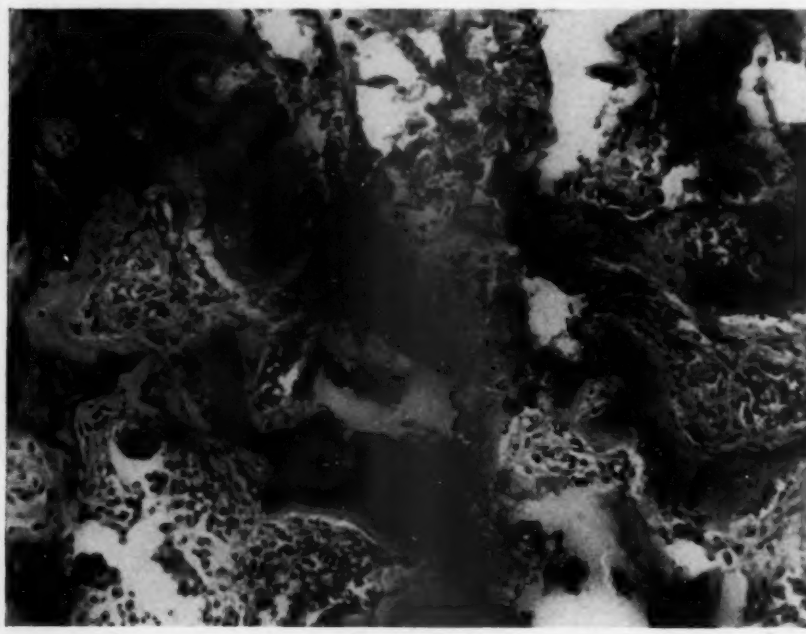

Fig. 176.—Photomicrograph showing bone changes in Paget's disease of the maxilla.

The postoperative course was uneventful, and the patient was discharged on Nov. 18, 1943. Her operative result was satisfactory, and the prognosis was favorable except that the bony enlargement might recur. Calcium metabolism studies are being continued relative to the generalized bone condition. The pathologic examination showed the type of bone changes seen in Paget's disease (Fig. 176).

Discussion.—The x-ray findings and the high phosphatase level point definitely to Paget's disease. Paget's disease frequently involves the upper jaw, but the mandible is very rarely affected. If teeth are present, they do not migrate as in acromegaly; the expansion of the maxilla occurs on the outside and inside of the dental arch. Hypercementosis, and resorptive processes on the teeth, repaired by apposition of Paget's type of bone is frequently seen in dentulous jaws.

VI. TUMORS AND CYSTS OF THE JAWS

Case 38

Fibro-Osteoma of Maxilla

N. E. (No. 420310), a 9-year-old boy, was seen on Sept. 9, 1943. He presented a large, lateral, noninflammatory swelling of the right maxilla, extending anteriorly to the right lateral incisor and involving the maxillary sinus. This growth had been steadily increasing in size for two years. The patient suffered no pain nor weight loss. A biopsy report of fibrous osteoma was made a year before. Six months before, the patient had received sixteen irradiation treatments with no improvement. There were no other significant findings in the physical history or examination.

Examination of the face and mouth revealed that the right cheek protruded further than the left. There was a swelling on the lateral surface of the right maxilla, extending down almost to the occlusal surfaces of the upper teeth (Fig. 177). A preoperative diagnosis of fibro-osteoma was made.

X-ray examination showed a cloudy, radiopaque mass in the right maxilla (Fig. 179), involving the entire maxillary sinus (Fig. 178).

On September 11 the patient was operated on for partial excision of the tumor, to obliterate the deformity and to obtain a biopsy specimen. The tumor was approached through an incision in the buccal mucosa under gas, oxygen, and ether anesthesia. The preoperative medication consisted of $\frac{1}{8}$ grain of morphine and $\frac{1}{200}$ grain of atropine one hour before operation. The incision was made along the gingival margin from the median line to the tuberosity, and was extended on each end vertically upward. The mucoperiosteum was detached with a periosteal elevator and retracted. The underlying bone was found to be soft, and a piece extending from the midline to the tuberosity, about $\frac{1}{2}$ inch thick, could be detached with an osteotome. Some of the bone which filled the maxillary sinus completely could be removed by means of a curette. The bone in general was very soft, was made up of finely honeycombed material, and could be carved easily. Bone was removed from the outer cortex as well as from the inside of the tumor. After dusting sulfanilamide powder into the wound, the mucoperiosteum was put into place, and held by interrupted silk sutures.

The pathologic diagnosis was ossifying fibroma. Microscopic examination showed newly formed, small, irregular bone trabeculae in fibrous connective tissue (Fig. 180).

Discussion.—This tumor has been described under various names, such as osteitis fibrosa localisata, osteodystrophy, osteofibroma, fibrous osteoma, or osteoid osteoma. The reason for so many different names is, in part, due to the varied appearance and changeable proportion of soft and hard tissue. Fibrous osteoma has been suggested by Phemister in an article published in the *Annals of Surgery* in 1937. In my book, *Oral Pathology,* I have used the term fibro-osteoma for a group of tumors with ossifying fibroma, a subdivision for types in which the fibrous part predominates. It is a slowly growing, benign bone tumor that grows without regard to anatomic landmarks. In the x-ray examination, a resemblance to Paget's disease is noted, especially in regard to the

Fig. 177.—Fibro-osteoma causing expansion of maxilla on right.

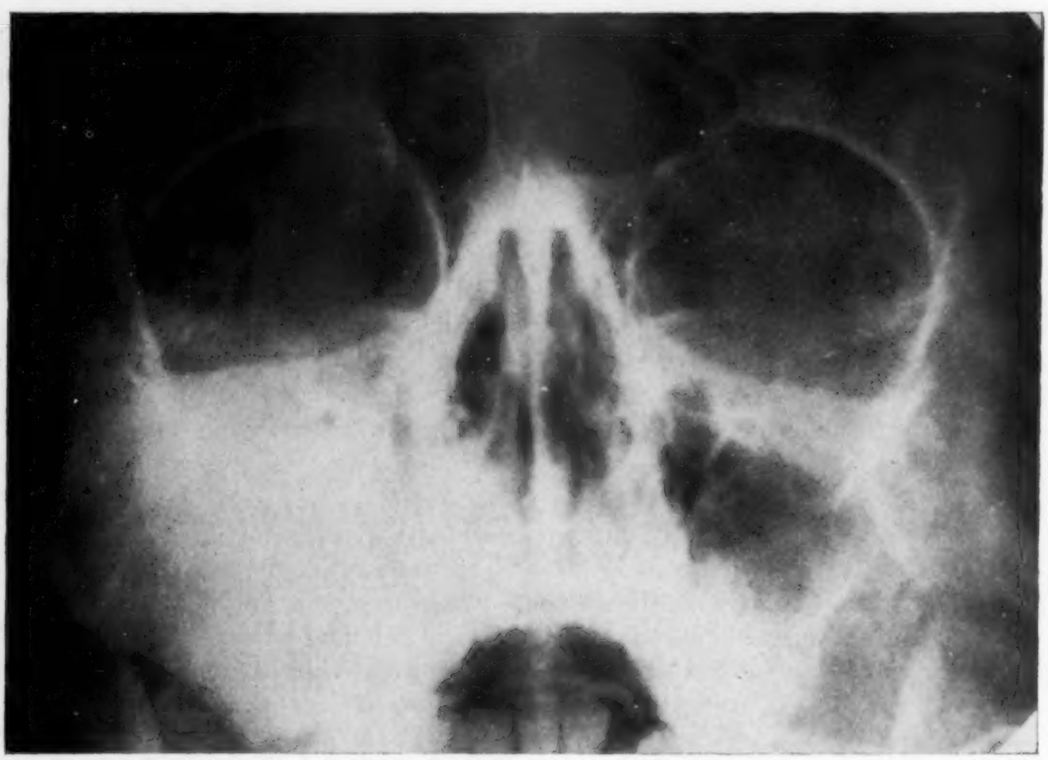

Fig. 178.—X-ray of fibro-osteoma of maxilla and malar bone involving maxillary sinus.

Fig. 179.—X-ray of fibro-osteoma of maxilla.

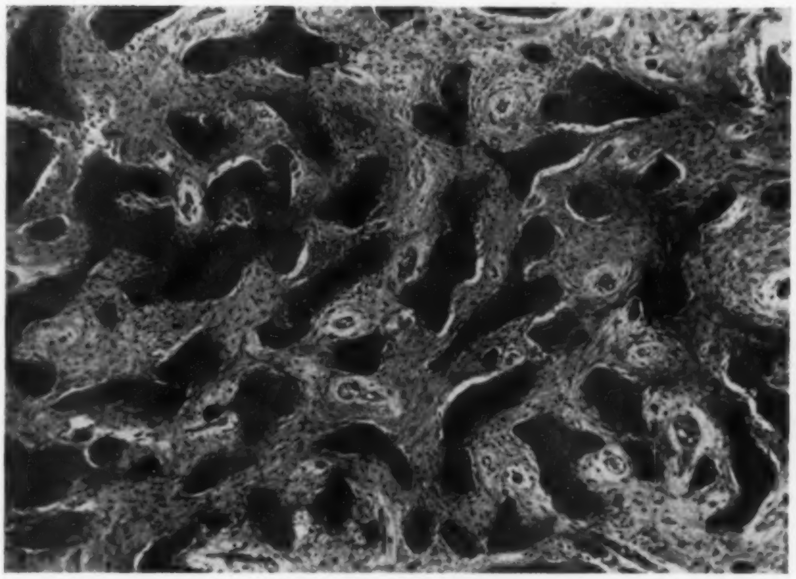

Fig. 180.—Photomicrograph of fibro-osteoma.

appearance of the maxillary sinuses. It generally progresses from the maxilla into the malar bone. Since it is not encapsulated or circumscribed, it cannot be excised completely. If the patient presents a deformity, part of the tumor may be removed, and irradiation in moderate amounts may be given with the hope that it may arrest further developments.

Case 39

Osteoma of Mandible

D. B. (No. 425412), a 28-year-old woman, came to the Outpatient Department on Oct. 21, 1943, complaining of a swelling over the ramus of the left mandible.

The mass had grown steadily for the past six or seven years. The only related cause to which she could attribute the swelling was the removal of an infected tooth in the molar region six months before the swelling appeared. The patient said that the mass pained her a little, and was slightly tender the first year she had it. She had noticed no change in the size during the last four or five years.

The past history was negative except for syphilitic infection in 1935. She was treated at the State Infirmary from 1935 until 1938, and has had negative blood tests twice a year since 1938.

Upon examination, a 2.5 by 2 cm. firm, hard mass was found on the inferior aspect of the ramus of the left mandible just anterior to the angle; it seemed to be fixed to the underlying bone. It was entirely asymptomatic. The patient was edentulous except for the lower incisors.

X-ray examination showed a homogeneous, dense mass, 3.5 cm. in diameter, projecting from the left mandible near the angle (Figs. 181 and 182). The report stated that "this could very well be a calcification in the submaxillary gland. If this is part of the mandible, it may have begun as a cementoma."

The patient was admitted to the House on Nov. 2, 1943, for excision of the osteoma of the jaw.

The preoperative medication was 1½ grains of nembutal at bedtime and at 8:00 A.M., and ⅙ grain of morphine and $\frac{1}{100}$ grain of atropine on call.

Under gas, oxygen, and ether anesthesia, a slightly curved incision was made at the angle of the jaw on the left, about 1 cm. beneath the inferior border (Fig. 183). The platysma was divided and the tumor exposed, which was covered by periosteum. This was incised and removed so that the attachment of the osteoma to the mandible became visible (Fig. 184). The attachment consisted of a fairly large pedicle about 1 by 1½ inches, which was connected with the inferior and lingual aspects of the bone. The pedicle was divided with an osteotome, and the tumor removed. Bone bleeding was stopped with adrenalin. The subcutaneous tissues were closed with catgut sutures. A rubber dam drain was inserted to take care of bleeding, and a subcuticular suture was used to close the skin (Fig. 185).

The pathologic report stated, "The gross findings showed an irregularly shaped, hard, bony mass, measuring 3.2 by 3.2 by 2.3 cm., and weighing 14 Gm.

Fig. 181.—X-ray of mandible showing osteoma.

Fig. 182.—X-ray of mandible in posteroanterior view showing osteoma extending below inferior border of jaw.

Fig. 183.

Fig. 184.

Fig. 185.

Fig. 183.—Submandibular incision.
Fig. 184.—Removal of excised tumor.
Fig. 185.—Subcuticular suture drawing the wound edges together.

The external surface is irregular, with areas of depression which are reddish, alternating with areas of white bone. On one side there is an area measuring 3 by 2 cm., where the surface is ragged and spongy, and apparently represents the site of removal of the tumor. The cut surface is hemorrhagic whitish-pink dense bone." Microscopic examination showed the entire tumor to be made up of cortical bone, such as is found in osteoma durum (Fig. 186).

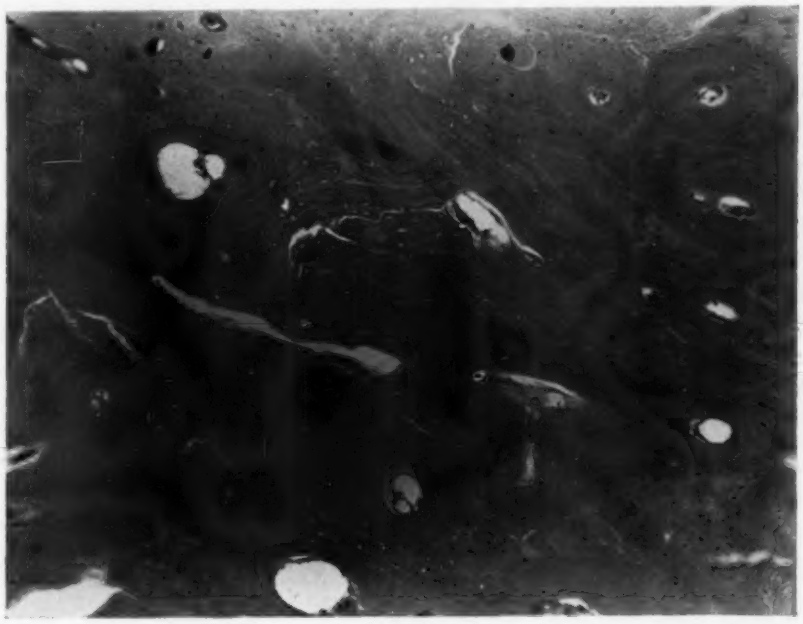

Fig. 186.—Photomicrograph showing tumor made up of cortical bone, as is found in osteoma durum.

Fig. 187.—X-ray taken after removal of osteoma. Note osteoblastic formations in various parts of jaw.

The postoperative course was uneventful and the patient was discharged on the fourth postoperative day to be followed in the Outpatient Department. She was next seen on Nov. 15, 1943, at which time there were no swelling nor signs of infection. The postoperative x-ray, shown in Fig. 187, shows the site where the pedicle of the tumor was detached from the jaws, as well as several osteoblastic areas inside. These are frequently seen in jaws bearing peripheral osteomas. The patient was in excellent condition. The suture was removed, and on Nov. 24, 1943, she was permanently discharged, with a final diagnosis of osteoma of the mandible.

Discussion.—This lesion was located principally on the outer surface of the mandible, but it extended below its inferior border to the lingual side. Because of its location and shape in the anteroposterior view, as well as in the lateral exposure, it might well be diagnosed as a calcified submaxillary gland. Osteomas are generally attached by a small pedicle to the surface of the jaw, and do not extend beneath it as was the case here.

Case 40

Odontogenic Cyst of Mandible

A. W. (413974), a 52-year-old woman, came to the Outpatient Clinic on July 26, 1943, complaining of a lesion on the left mandible. The patient had had all her teeth removed about two years before, and had been fitted with upper and lower dentures, which she had worn constantly until she broke the lower set about three weeks before admission. Shortly thereafter she noted a gradual swelling in the molar region. She had little or no pain from this but felt some tenderness when eating. The lesion rapidly expanded.

Examination showed a cystic mass projecting about 2 cm. over the surface, involving the lower left mandible from the second molar area to the canine region. The mass was smooth and not painful, but fluctuant. There was some greenish discoloration of the mucosa (Fig. 188). No submaxillary or cervical lymph nodes were palpable.

X-rays were taken on July 26, 1943, which showed a large rounded cystic area involving the left mandible, measuring about 2 inches in length. Its edges were smooth. The mandible was almost divided at one point, and the cortex at no place was thicker than 3 mm. (Fig. 189). The findings were those of a large cyst of the jaw.

On July 28, the patient was admitted to the House for removal of the cyst. The operation was performed, on July 29, with nitrous oxide, oxygen, and ether anesthesia given by the endotracheal method. The preoperative medication consisted of $\frac{1}{6}$ grain of morphine sulfate and $\frac{1}{100}$ grain of atropine on call. An incision was made on the surface of the swelling, after which the mucoperiosteum was carefully dissected away from the cyst wall. The thin layer of bone, which had a bluish color, was excised, after which a large amount of cystic fluid escaped into the mouth. It was of a yellowish color, containing cholesterin crystals. The cyst sac was then dissected out by means of a periosteal elevator, and removed. The mucosal flap was placed into the bottom of the cyst cavity and held there by means of boric strips.

The pathologic diagnosis was odontogenic cyst. The specimen was made up of thin fibrous tissue lined on one surface by a glistening membrane, and including a small piece of bone. The section also included a large nerve.

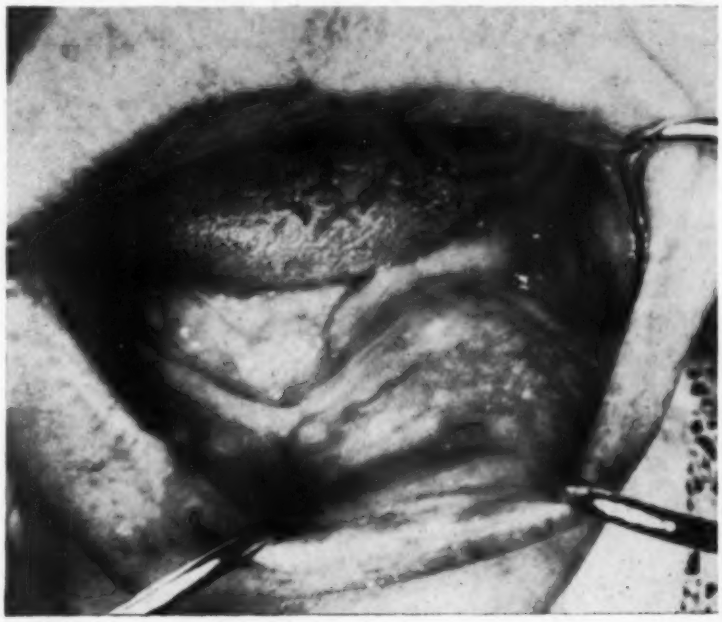

Fig. 188.—Photograph of swelling caused by mandibular cyst.

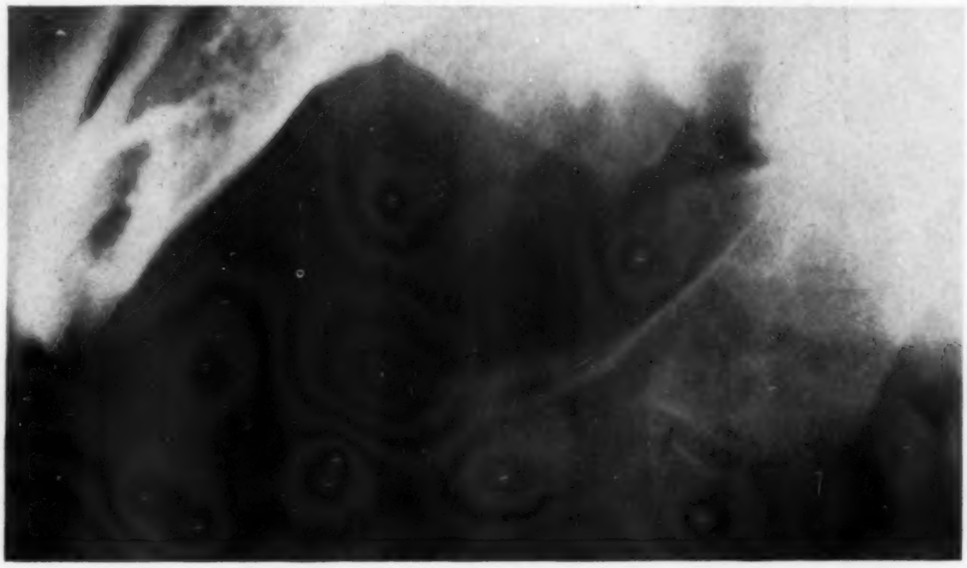

Fig. 189.—X-ray showing cystic defect in mandible and thinned out, expanded bone.

On the first postoperative day there was marked edema of the floor of the mouth. The temperature was 100° F., but the physical examination was negative.

The patient recovered well and was discharged to the Outpatient Department for follow-up treatment on the fourth postoperative day. By Aug. 23,

1943, the cyst cavity had become lined by an epitheliated granulation tissue, so that the dressing could be discontinued. The patient was advised to irrigate the cavity after each meal. She was last seen on Oct. 1, 1943, when she was advised to continue treatment at home until the cavity is completely obliterated.

Case 41

Odontogenic Cyst of Maxilla

L. C. (No. 491843), a 48-year-old man, was referred to the hospital on Sept. 3, 1943, with the chief complaint of a lump on the upper right jaw.

For a year, the patient had noticed the lump, which extended from the alveolar process to the palate, and which was irritated by his denture. Recently, the lump had grown larger, and there was some pain in this area. There was no bleeding. X-rays were taken by his doctor, who advised the surgical removal of the lump.

Fig. 190.—Odontogenic cyst of maxilla.

Roentgen examination made previous to admission showed an osteolytic area extending from the canine region to the tuberosity. It had an indistinct margin such as is frequently seen in infiltrative tumors (Fig. 191).

Examination of the mouth showed it to be edentulous. On the right side of the maxilla, a swelling which obliterated the bucco-alveolar sulcus could be seen. It was bluish green at the most prominent part, and slightly fluctuant (Fig. 190); it had the appearance of a cyst filled with fluid, but the x-ray evidence was not in agreement with such a diagnosis.

An operation for the excision of the tumor was performed on Sept. 4, 1943, under intravenous pentothal sodium anesthesia. The preoperative medication was $1\frac{1}{2}$ grains of nembutal at bedtime, 3 grains of nembutal three hours before operation, and $\frac{1}{4}$ grain of morphine, $\frac{1}{200}$ grain of scopolamine, and $\frac{1}{120}$ grain of atropine two hours before operation. An incision was made over the surface

of the tumor, and, after dissecting the mucoperiosteum, it was found that the tumor was a cyst. The cyst membrane was detached from the oral mucosa and the bone, by means of a periosteal elevator, and removed in toto. A large cavity was visible, lined by cortical bone. The bleeding was arrested by means of adrenalin packs. The mucoperiosteal flap was placed into the cavity, and held by means of a boric strip pack.

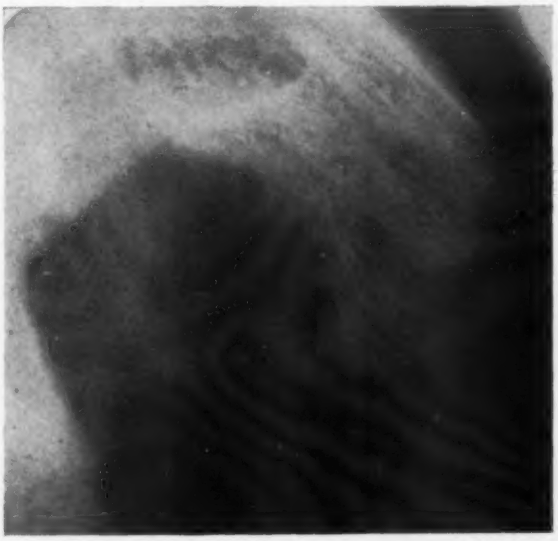

Fig. 191.—Odontogenic cyst of maxilla.

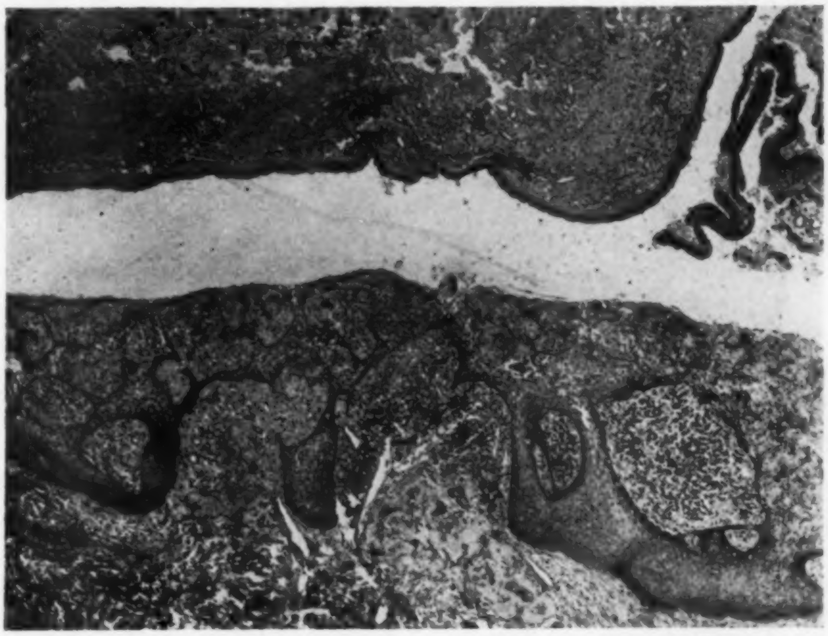

Fig. 192.—Photomicrograph showing section of cyst with epithelial lining and inflammatory infiltration.

The pathologic report classified the tumor as a cyst, probably radicular in nature. The cyst was lined by a well-differentiated squamous epithelium with an underlying, marked inflammatory infiltration (Fig. 192).

The patient's postoperative course was uneventful, and he was discharged from the hospital on the second postoperative day; he was advised to report for dressings every two or three days.

Discussion.—In this case, the x-ray findings were misleading. This shows the importance of the clinical findings, which are so frequently overlooked today because we have come to rely on the x-ray as an almost unfailing diagnostic medium.

Case 42

Globulomaxillary Cyst

E. S. (No. 424239), a 17-year-old girl, was admitted on Oct. 14, 1943, because of a swelling on the left side of the face.

The patient had been well until three months before admission, when she noted the onset of a progressive, painless swelling of the left maxillary region. This appeared without preceding trauma, dental extraction at that site, or respiratory infection. She had no headaches nor visual disturbances, and there was no pain on mastication. The swelling continued without ever showing heat or redness, until eight days before admission to the hospital. At that time the patient noticed a lump in the left upper gingiva, approximately over the canine tooth. This increased in size and was painless. Five days before admission her local dentist incised the lump. Considerable white material was discharged, and the patient said that there was no taste to this material. Both the intraoral and the maxillary swelling immediately decreased in size. An x-ray taken by her dentist showed a cystic area and an impacted tooth.

Examination of the patient showed a swelling starting between the canine and second incisor tooth, and extending over the canine fossa. There was evidence of a previously made incision and slight fluctuation. The second incisor was carious, and the canine contained a filling in the distal side. The patient also presented a hypertrophied labial frenum.

X-ray examination, on Oct. 14, 1943, showed a cystic lesion in the left maxilla extending from the central incisor to the molar area. An unerupted malposed right upper canine was found, but there was no definite evidence of the tooth being within the cyst (Fig. 193). The findings were those of a cystic lesion, with a question of its being an adamantinoma.

In reviewing the x-rays, the lesion was classified as a facial cleft cyst or fissural cyst. It was distinguished from an odontogenic cyst by the fact that it extended between the second incisor and canine tooth, causing divergence of the roots (Fig. 194). Such cysts, being formed from epithelium enclaved between the globular and maxillary embryonic processes, are specifically globulomaxillary cysts.

The patient was admitted to the House, and, on November 15, was operated on under gas, oxygen, and ether anesthesia. The preoperative medication was $\frac{1}{8}$ grain of morphine, $\frac{1}{150}$ grain of atropine on call. An incision was made on the outer surface of the maxilla about $1\frac{1}{2}$ inches in length. The mucoperiosteum was dissected away, and a bulging cyst wall exposed. The bony wall of the cyst was removed, after which a thick cyst sac was dissected out. The cyst cavity was about 1 inch in diameter, and had encroached upon the antral

cavity. The membrane of the maxillary sinus was visible, but was not perforated. Sulfanilamide was dusted into the wound, and the mucoperiosteal flap was placed into the bottom of the cavity; a boric strip was inserted. The hypertrophied labial frenum was then excised and two sutures taken to close the wound.

The unerupted right maxillary canine was next exposed by an incision made on the palate along the alveolar border, and the mucosa retracted. The bone

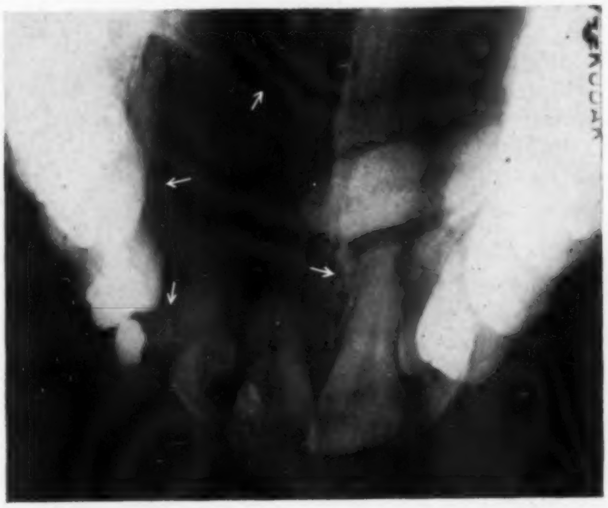

Fig. 193.—Globulomaxillary cyst on left with unerupted canine on right.

Fig. 194.—X-ray of globulomaxillary cyst showing cystic area causing the roots of the maxillary canine and second incisor to diverge.

was cut away with chisels, and the tooth loosened with elevators, and removed. Sulfanilamide powder was dusted into the wound, and it was closed with silk sutures.

The pathologic diagnosis was cyst of the maxilla with chronic inflammation.

The postoperative course was uneventful, and the patient was discharged on the first postoperative day, to be followed in the Outpatient Department.

Discussion.—In making a diagnosis in this case, three theories might be developed: (1) That the cyst is a dentigerous cyst because the canine looks as if it extended into the cystic cavity. Cysts frequently form from the enamel organ of unerupted teeth, but generally they surround the entire crown of the tooth. (2) That the cyst is a globulomaxillary cyst. The divergence of the canine and lateral incisor is a typical sign of such cysts. They start developing between the roots of these teeth and press them apart. Later they grow upward into the bone. (3) That the cyst is a radicular cyst, formed from a granuloma which might have developed on the carious incisor. Such cysts do not grow between the roots of two teeth; they form a circular area around the apex of the tooth in question. However, it is quite likely that the carious process in the incisor caused an ascending pulpitis in this tooth, and, via its apical foramen, infected the globulomaxillary cyst, which would nicely account for the sudden increase in the swelling over the maxillary part of the face.

Case 43

Adamanto-Odontoma

G. S. (No. 430131), a 35-year-old housewife, was referred to the Massachusetts General Hospital by her local physician, who took x-rays of her left jaw and made a diagnosis of tumor.

The patient had had a painless swelling of the left jaw for about a year and a half, which caused a marked deformity of her face (Fig. 195). This mass had gradually increased in size without ever having caused pain, limitation of jaw motion, or inconvenience of any kind.

Examination showed a firm, bony mass over the left ramus of the mandible, which was not movable. The motion of the mandible was not limited. A crackling sensation on palpation was felt.

Roentgen examination revealed a large cystic cavity involving the entire left ramus and part of the mandible. A molar tooth and other odontogenic structures were included in the molar region (Fig. 196). The anteroposterior view revealed marked expansion of the ramus on its inner and outer surfaces (Fig. 197). A roentgen diagnosis of cystic odontoma was made, and an operation for the removal of the cyst was advised.

The operation was performed on Dec. 7, 1943, under nitrous oxide, oxygen, and ether anesthesia. The preoperative medication consisted of $1\frac{1}{2}$ grains of nembutal at bedtime, and 5 grains of sodium luminal two hours before operation. On call to the operating room, $1\frac{1}{2}$ grains of nembutal, $\frac{1}{6}$ grain of morphine sulfate, and $\frac{1}{100}$ grain of atropine were given. After the usual preparation of the field of operation, an incision was made intraorally along the entire anterior

border of the ramus, and continued along the alveolar crest of the mandible. The mucoperiosteum was detached and the bone exposed. There was a perforation in the molar triangle of the ramus, through which a great deal of cystic fluid escaped. The cystic cavity was apparent. The cyst sac was gradually dissected away from the bone, and grasped with forceps. In some places it could be easily shelled out; in others it was firmly attached to the bone. It seemed to consist of two parts: one in the posterior part of the ramus and angle of the jaw, and the other in the mandible around the displaced tooth. The posterior cyst sac was removed first. It seemed to contain in various places mural thickenings, and when closely examined, a number of small, rudimentary teeth could be seen projecting through its surface. The anterior cyst was enucleated next. This cyst had formed around the unerupted molar tooth, which was loosened with elevators, and removed with its cyst sac. After the bone was somewhat reduced with rongeur forceps to widen the opening, the entire cavity was packed with a large packing of several strips of boric iodoform gauze.

Fig. 195.—Patient with adamanto-odontoma causing expansion of ramus.

The pathologic specimen presented the following gross appearance: "An irregular reddish-pink smooth sheet of membranous tissue and attached fibrous tissue measuring 6 by 2 by 0.2 cm. Embedded in the fibrous tissue are several firm, white, smooth, encapsulated nodules, varying from 0.3 to 0.8 cm. in diameter. Attached to one corner of the sheet is an irregular reddish-pink, mucosa-covered mass of firm tissue, 1.5 cm. in diameter. Studded over the surface of the nodule are many pearly-white, round, smooth, calcified nodules varying from 0.2 to 0.4 cm. in diameter. Embedded in the center is an abortive tooth root."

Fig. 196.—X-ray shows the cystic area including a molar and many odontogenic structures.

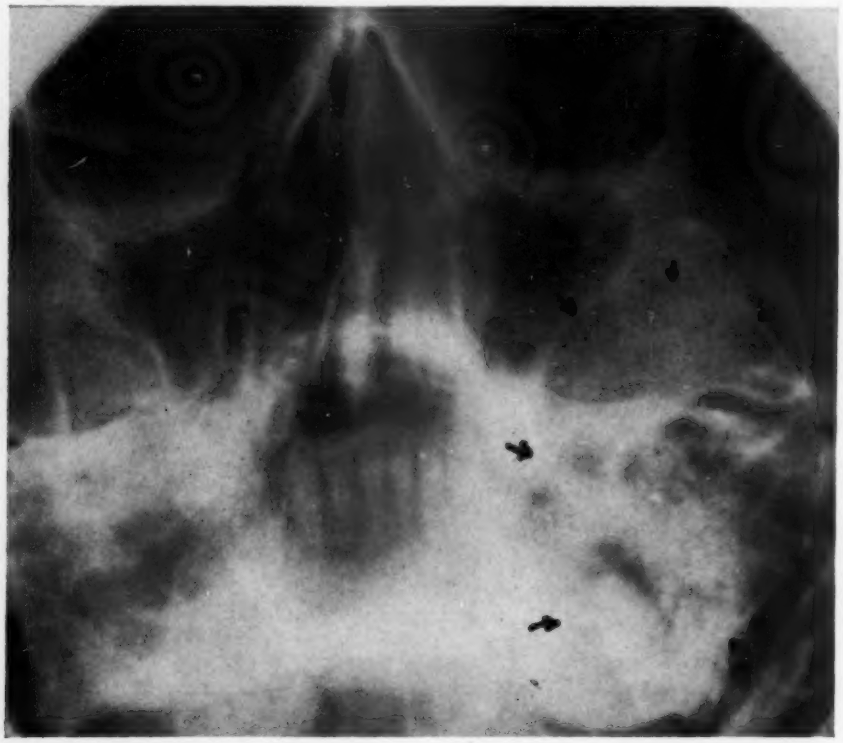

Fig. 197.—Posteroanterior view showing expansion of inner and outer sides of the ramus.

Fig. 198.—Photomicrograph showing abortive tooth root and sections through three small rudimentary teeth. *E*, Enamel space, the one in the middle containing a core of dentine, the one on the right containing dentine with pulp canal. Note arrows pointing to proliferating epithelium given off from its epithelial membrane.

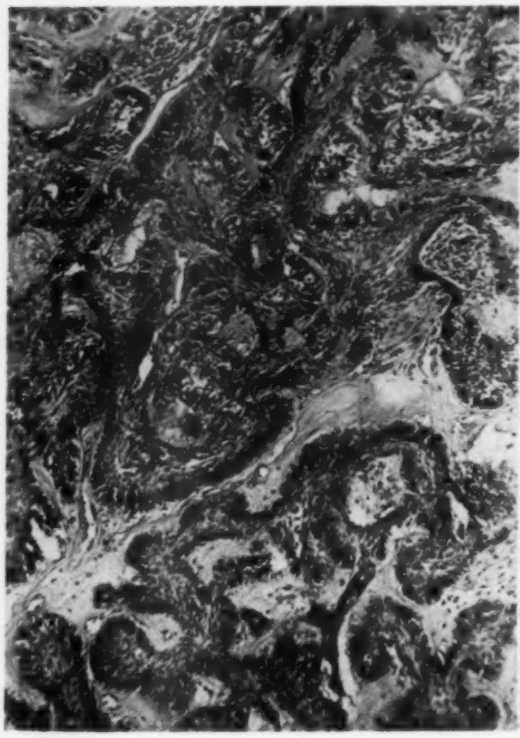

Fig. 199.

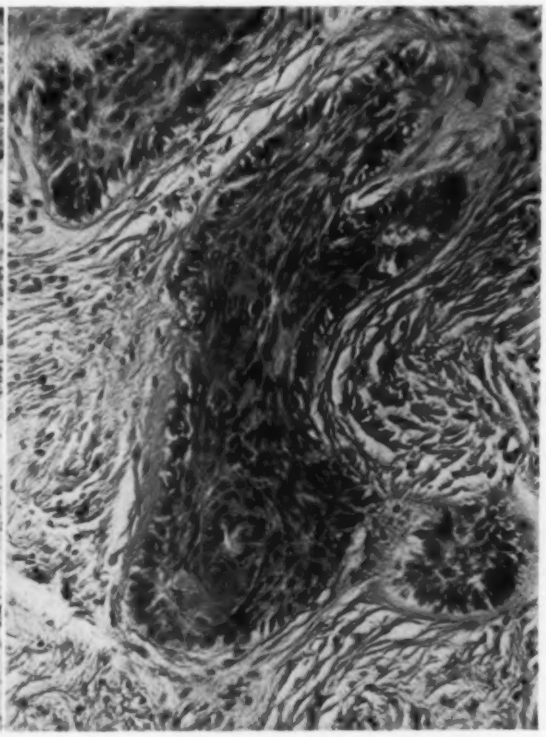

Fig. 200.

Fig. 199.—Irregular follicles of enamel epithelium.

Fig. 200.—Single follicle in higher magnification showing pseudo pearls.

Microscopic examination showed a large calcified mass (described as the abortive tooth root), which is made up of irregularly formed dentine and lined by cementum which forms various projections. Close to this mass were found sections through small rudimentary teeth. These were made up of an epithelial membrane surrounding spaces which represented the enamel of these teeth that dissolved away during decalcification of the specimen. Some of the more immature enamel was retained and was attached to a core of dentine which might or might not show a central pulp canal. The epithelial membrane of these structures in some instances was surrounded by connective tissue. Occasionally, cementum extended from the tooth and connected with it. At one place (marked by an arrow), epithelial structures were given off, and proliferated into the adjoining tissue (Fig. 198). Another specimen (taken from the cyst in the ramus) showed proliferation of epithelium which developed in the form of irregular follicles surrounded by connective tissue (Fig. 199). These were made up of an outer layer of cylindrical cells and contained indifferent cells, some of which showed the tendency to develop into squamous cells and form pseudo pearls (Fig. 200). Diagnosis: odontoma and adamantoblastoma combined, viz., odonto-adamantoblastoma of mandible.

The postoperative course was uneventful, and the patient was discharged on the second postoperative day, to be followed in the Outpatient Department.

Discussion.—Apparently, we are dealing here with a dentigerous cyst formed from the enamel organ of the unerupted second molar, as well as a cystic lesion formed from the aberrant third molar. Here, the tooth germ developed into an odontoma forming numerous particles of calcified tooth substance, but, in addition, the epithelial constituents started to proliferate and formed an adamantoblastoma. Thus, the rare combination of odontoma and adamantoblastoma came into existence. The adamantoblastoma does not, as a rule, produce calcified tissue; it is a tumor formed by the overgrowth of enamel epithelium and is generally present in the form of follicles containing cells in the various stages of development. It should be noted that the epithelium in this case is almost entirely made up of preameloblastic cells; stellate and cuboidal forms are rare.

Case 44

Adamantoblastoma of Mandible

H. C. (No. 420291), a 47-year-old man, was admitted to the hospital on Sept. 9, 1943, because of a tumor on the lower alveolar ridge.

For four years the patient had had a firm, growing mass between the lower left incisors and the lip. This mass had displaced several teeth. Otherwise, it had been painless and asymptomatic.

Examination of the mouth revealed a firm, round swelling extending from the midline over to the left on the anterior surface of the lower alveolar process, and pushing the teeth slightly backward, separating the canine and the second incisor teeth (Fig. 201). The only significant factor brought out in the physical examination was a question of mild chronic bronchitis.

X-ray examination revealed a small polycystic area between the canine and second incisor, which have been forced apart. The lesion involved only slightly

the subapical area, and there was no evidence of infiltration of the surrounding bone by the tumor (Fig. 202). A roentgen diagnosis of central benign giant-cell tumor was made by the roentgenologist in a hospital from which the patient had been referred to this clinic.

Fig. 201.—Adamantoblastoma expanding alveolar process in anterior part of mandible.

Fig. 202.—X-ray film showing polycystic central tumor.

On Sept. 10, 1943, an operation was performed for the excision of the tumor of the gingiva and mandible. The preoperative medication consisted of 1½ grains of nembutal at bedtime, and $\frac{1}{6}$ grain of morphine with $\frac{1}{100}$ grain of atropine one hour before operation. The anesthetic used was endotracheal gas, oxygen, and ether, supplemented with local injections of monocaine epinephrine to decrease hemorrhage. An incision was made around the tumor on the outer surface, as well as on the inner surface of the mandible. With a chisel, a block containing the left lower canine and the left lower incisor was detached on each side and below, and removed with the tumor attached. The bone hemorrhage was arrested with adrenalin, and the tumor bed treated with Carnoy's solution. A boric strip pack was then applied, and held down with silk sutures taken in the edges of the soft tissue wound.

The pathologic specimen was described as "a mass 1.5 by 1.5 by 1 cm., imbedded in which were two teeth. On section through the gingiva between the roots of the teeth, a 1 cm. pinkish grey, somewhat encapsulated nodule of soft glistening tissue was exposed." Sections were cut in a mesiodistal plane to show the tumor tissue between the teeth. The tumor has caused resorptive areas where it was in contact with the tooth surfaces. In the incisor, the

Fig. 203.—Adamantoblastoma forming between canine and second incisor teeth, causing resorption of tooth tissue.

Fig. 204.—Adamantoblastoma, higher magnification, showing epithelial follicles containing cysts or stellate and squamous cells.

cementum from the apex to the transeptal fibers has been lost; the canine has a smaller area involved, but the resorptive process extended deep into the dentine (Fig. 203). The soft tissue mass was composed of epithelial follicles lined by cylindrical cells, and surrounded by a connective tissue stroma. Many of the follicles contained small cysts, while others were filled with stellate and squamous cells (Fig. 204). The diagnosis of adamantoblastoma was made.

On the first postoperative day, the patient's temperature rose to 101° F. This was due to slight atelectasis and chronic bronchitis.

The postoperative course was uneventful, and the patient was discharged on Sept. 13, 1943. In February, 1944, the patient was re-examined and there was no evidence of recurrence.

Discussion.—This is an illustration of an adamantoblastoma developing late in life; therefore, it must have formed from epithelial cell rests in the periodontal membrane of the involved tooth or teeth.

Case 45

Adenocarcinoma of Mandible

R. H. (No 430658), a 67-year-old widow, entered the hospital on Dec. 6, 1943, with a chief complaint of a swelling of the left jaw.

For many years this patient had been conscious of a wisdom tooth in her left lower jaw which pained her occasionally. Ten months before she entered the hospital, her left jaw began to swell gradually, and to become painful. This was thought to be due to the tooth, which had been removed eight months before. The condition however progressed until the patient was unable to open her mouth more than an inch. She was unable to take food well, and had lived on a fluid diet for the most part. At the onset she had the "flu," and ever since that time she had had a cough, and had raised thick mucus daily, but no blood. During this ten-month period she had lost 60 pounds. Her appetite had been good.

The general health of the patient was fair. The system review was essentially negative except for a question of heart enlargement to the left. She had an apical systolic murmur which was not transmitted, and a systolic murmur at the aortic area. Her blood pressure was 160/100. The patient was considered a poor anesthetic risk.

Examination showed a hard swelling of the side of the left jaw. No regional glands were felt. There was a left facial paralysis (Fig. 205). Intraoral examination showed obliteration of the buccomandibular fold by a firm swelling attached to the jaw. It had a greenish color, with blood vessels clearly outlined under the epithelium of the mucosa (Fig. 206).

The anesthesia consultant suggested that since the patient could not open her mouth, intubation with pentothal sodium would be difficult. For this reason, and because of her hypertension and questionable cardiac status, intratracheal nitrous oxide, oxygen, and ether with a nasal tube and good pharyngeal packing was advised.

Roentgen examination of the mandible showed a destructive process, which had the appearance of a metastatic tumor rather than a primary one (Fig. 207).

Chest x-rays were taken on the day of admission. The lung fields were clear throughout. The diaphragm was smooth, and showed a normal excursion. The heart was not remarkable. The aorta was slightly tortuous and showed

calcification of its arch. There was no evidence of metastases to the lungs or bones of the thoracic cage.

On Dec. 7, 1943, a biopsy of the tumor of the jaw was performed under intratracheal nitrous oxide, oxygen, and ether. The preoperative medication

Fig. 205.—Photograph showing swelling of face and facial paralysis caused by adenocarcinoma.

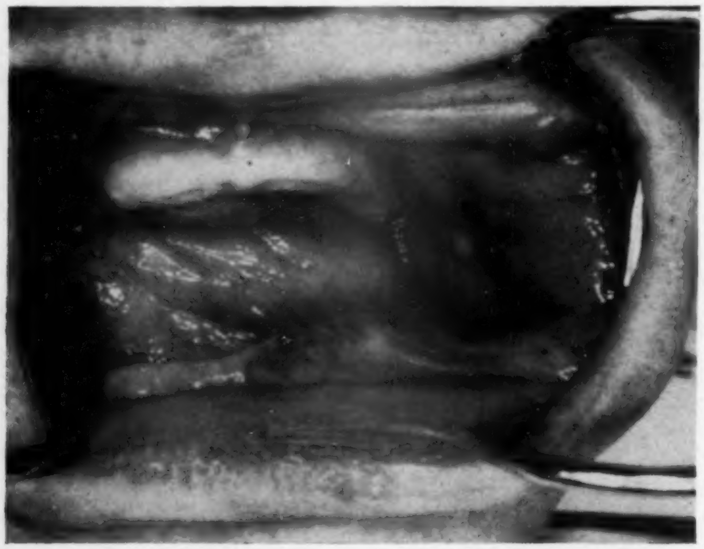

Fig. 206.—Intraoral swelling of jaw caused by adenocarcinoma.

consisted of $1\frac{1}{2}$ grains of nembutal at bedtime, and $\frac{1}{6}$ grain of morphine and $\frac{1}{100}$ grain of atropine on call to the operating room. The anesthesia was discontinued when the operation was begun. The oropharynx was packed with wet gauze, and wet gauze was placed over the nostrils. Then, with the

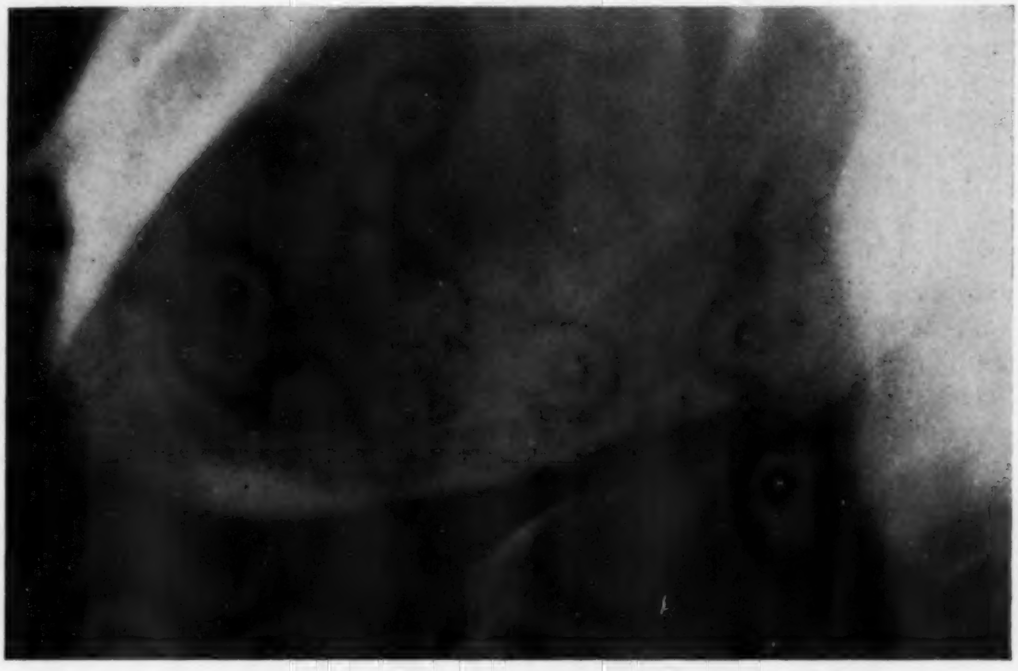

Fig. 207.—Metastatic adenocarcinoma of mandible.

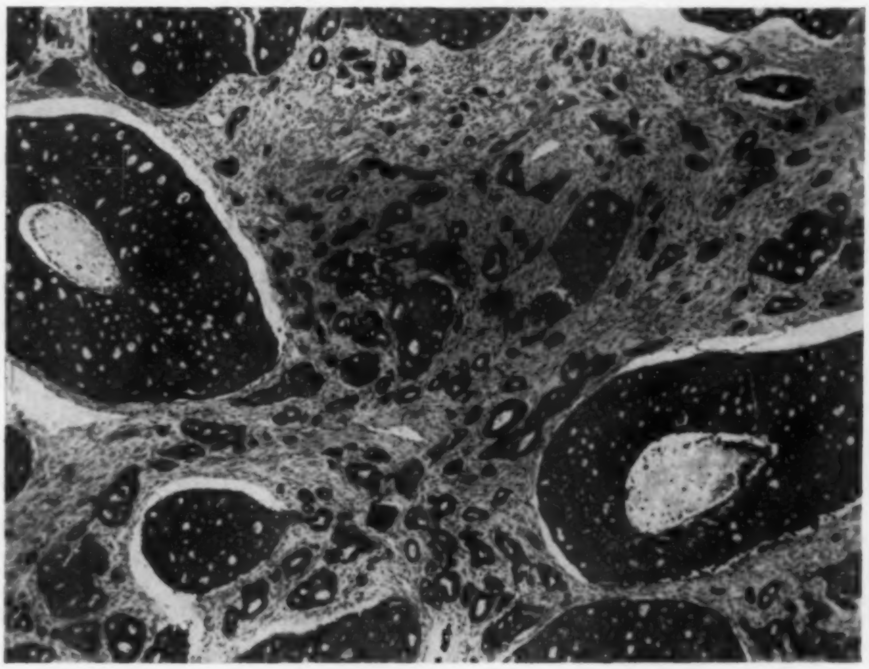

Fig. 208.—Photomicrograph of adenocarcinoma of mandible.

diathermy knife, an incision was made on the middle of the tumor down to the bone, and, by means of an osteotome, a piece of bone was detached. The wound was closed with sutures after cauterization. Following the surgical procedure, the patient was given an intravenous infusion of 1,000 c.c. of 5 per cent dextrose in saline solution.

The pathologic diagnosis was adenocarcinoma. The specimen consisted of the following: "Two pieces of tissue, which are reddish-white, moderately firm, partially encapsulated, measuring, respectively, 1.5 by 0.5 by 0.1 cm., and 1.5 by 1 by 0.5 cm. One border of the larger piece contains a small area of reddish-brown cancellous bone intimately connected with the surrounding tissue. The cut surface of both pieces are grayish-white, granular, and dotted with small, yellow granules measuring 1 to 2 mm. in diameter." The tumor was not suggestive of a metastatic lesion from a distant primary source. It probably arose from the glands in the mouth.

The postoperative course was smooth, and the patient was discharged on the fifth postoperative day to her local doctor. Her condition was not improved, and she was advised to have palliative radiotherapy with the hope of arresting the growth, and perhaps of improving her facial paralysis.

Discussion.—This patient has, undoubtedly, a malignant tumor of the jaw. Since facial paralysis had developed, it was felt that a small malignant tumor in the parotid gland might be the primary lesion, which is in agreement with the findings and opinion of the pathologist.

VII. DEFORMITIES OF THE JAWS

Case 46

Ankylosis Associated With Micrognathia, Corrected by Osteoarthrotomy and Unilateral Sliding Osteotomy

R. F. (No. 413232), a 23-year-old man, was admitted to the hospital with a chief complaint of difficulty in opening his jaw.

This patient had had a birth weight of 14 pounds. During a breech delivery, his wrist was broken, and there was a fracture of the left leg. At this time, a jaw injury probably occurred. Soon after birth it was noticed that the patient could open his mouth only slightly. The injury resulted in ankylosis, and an under-development of the lower jaw (Fig. 209).

In 1934, the patient was seen in the Outpatient Department, and it was thought that he was too young for plastic surgery. The x-ray report for March 4, 1934, stated that "there is deformity of the lower jaw apparently due to underdevelopment of the horizontal ramus on both sides. The left molars are unerupted and malposed, and there are multiple carious teeth present."

In May, 1943, he was seen by an exodontist, who extracted all his teeth because they were so decayed and abscessed (Figs. 210 and 211) that they could not be filled.

In July, examination of the mouth showed ankylosis of the mandible with marked micrognathia of the lower jaw due to a unilateral underdevelopment. The jaw was concave on the right, and the median line of the mandible was dis-

placed about 1 inch to the left. The movements were limited to about 2 mm. vertically, and 5 mm. in lateral excursion. The mouth was edentulous, and the median line of the mandible was displaced to the left (Fig. 212). The tonsils and posterior pharynx could not be seen because of the patient's inability to open his jaws.

Fig. 209.—Micrognathia due to unilateral ankylosis of the jaw.

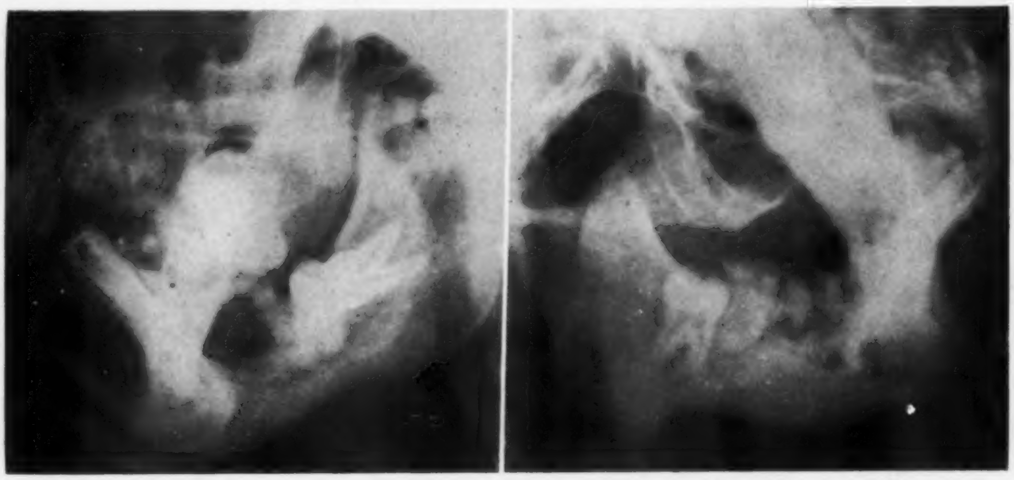

Fig. 210.—X-ray of left mandible showing carious teeth and unerupted molar.
Fig. 211.—X-ray of right mandible showing carious abscessed teeth and unerupted molar.

Examination of the neck showed the right sternocleidomastoid muscle to be hard and fibrosed. The neck was fixed laterally, slightly to the right, and the head rotated slightly to the left.

X-ray examination showed a hyperostosis of the left condyle, which probably accounted for the ankylosis (Fig. 213). The left mandible was shorter than the right.

An osteoarthrotomy was performed on the patient on July 22, 1943. The preoperative medication was nembutal, 1½ grains, at bedtime, and 1½ grains two hours before operation with $\frac{1}{300}$ grain of scopolamine. One hour before the operation the scopolamine was repeated, and $\frac{1}{6}$ grain of morphine and $\frac{1}{120}$ grain of atropine were administered. The anesthetic agent was intravenous pentothal sodium. An angulated vertical incision was made (Fig. 214) in front of the left ear, and the fascia divided. The transverse facial artery and vein were ligated and cut, and the zygomatic arch exposed. A very much enlarged, hypertrophied condyle was located. The capsule over this condyle was incised, as well as the periosteum at the neck of the condyle. An osteotomy was performed in the subcondylar area by means of burrs and chisel (Fig. 215). A chisel was then inserted into the articular space and the condyle pried loose from the glenoid fossa. After detaching the remainder of the capsule, the condyle was removed. Sulfanilamide powder was inserted in the wound after the hemorrhage was arrested by ligating two or three muscular vessels. The fascia was closed with catgut sutures, and a rubber dam drain inserted in the wound. The incision was closed with subcuticular sutures (Fig. 216). A pressure pack and bandage were applied.

Postoperatively the patient was given an intravenous injection of 1,500 c.c. of 5 per cent saline and glucose. The postoperative course was uneventful, and the patient was discharged on the sixth postoperative day. The mobility of the jaw was improved and exercises prescribed to be carried out at home. The appearance of the incision six weeks after the operation is shown in Fig. 217.

The second admission to the hospital was on Sept. 24, 1943, for the next stage of treatment, which was diagonal osteotomy and advancement (Fig. 218). The preoperative medication consisted of 1½ grains of nembutal at bedtime and again the next morning; $\frac{1}{200}$ grain of scopolamine, $\frac{1}{6}$ grain of morphine, and $\frac{1}{120}$ grain of atropine were given just before operation. On September 25, under intravenous pentothal sodium anesthesia, an intraoral incision was made on the left side of the mandible from the ramus to the midline, with vertical extensions on the outside of the jaw at each end. The mucoperiosteum was laid back and the external surface of the jaw exposed. The mental foramen was located, and found to be in the region of the second molar. By means of surgical drills, a vertical cut was made in the posterior end of the bone, and a diagonal cut was outlined with drill holes extending toward the region of the chin (Fig. 219). The osteotomy was then completed by a vertical extension at the anterior end, through the inferior border of the jaw. The drill holes were connected, and the jaw was divided *without* the use of a chisel. The drill was constantly cooled by a saline drip, with a suction tube in the posterior part of the mouth to aspirate the fluid and blood (Fig. 220).

When pulling the fragments apart, a great deal of resistance was encountered from the mylohyoid muscle and the mucosa of the floor of the mouth. The tissue was divided, which allowed the anterior fragments to be advanced.

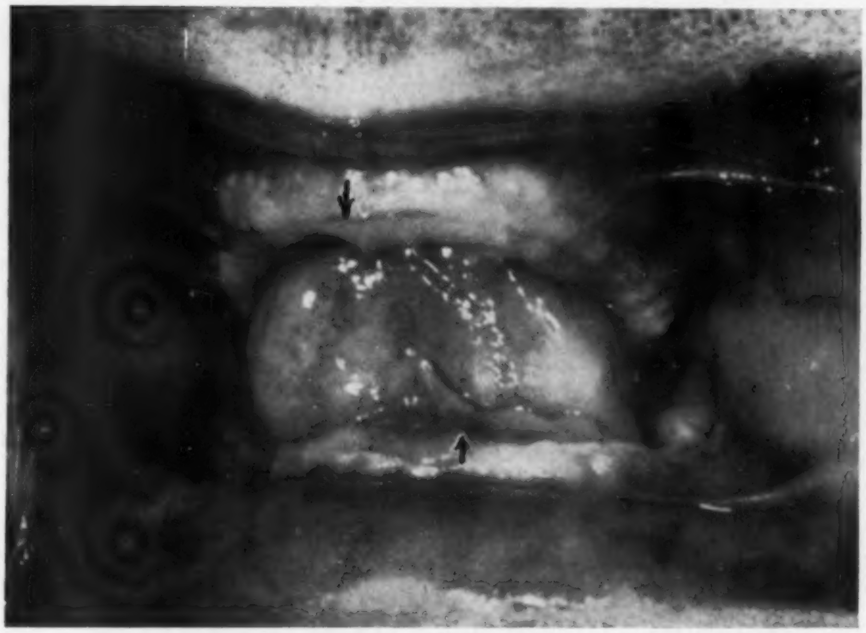

Fig. 212.—Edentulous jaws; median line of lower jaw is off-center.

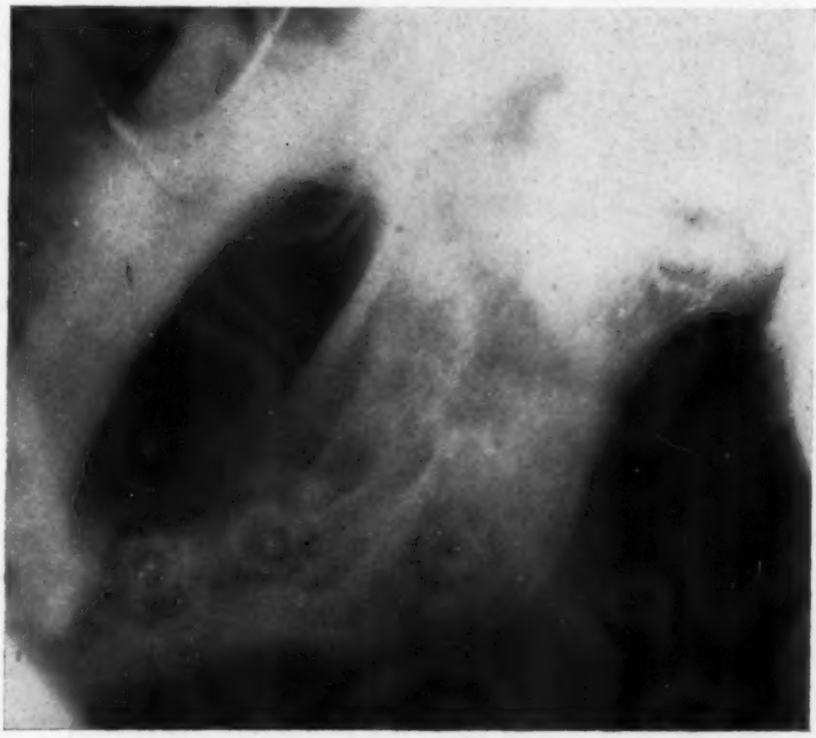

Fig. 213.—X-ray showing deformed condyle of mandible at the site of ankylosis.

Fig. 214.—Vertical incision for osteoarthrotomy.

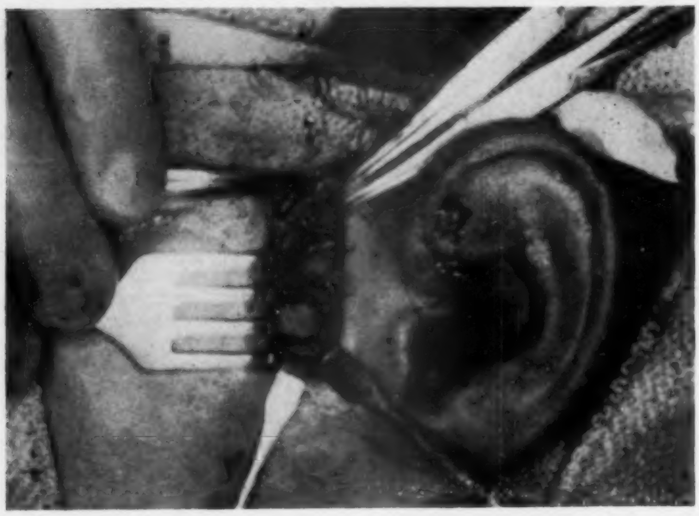

Fig. 215.—Zygomatic arch and condyle exposed; osteotomy in subcondylar area separates condyle from ramus.

Fig. 216.—Wound closed with subcuticular suture. Rubber dam drain inserted for twenty-four hours to prevent hematoma.

A drill hole was then made in the anterior part of the posterior fragment, and a notch in the superior part of the anterior fragment. Tantalum wire was inserted through the hole and tied over the notch, which united firmly the two fragments of bone. Sulfanilamide powder was dusted into the bone wound, the mucosa was replaced and attached by interrupted silk sutures.

Fig. 217.—Scar in front of ear six weeks after operation.

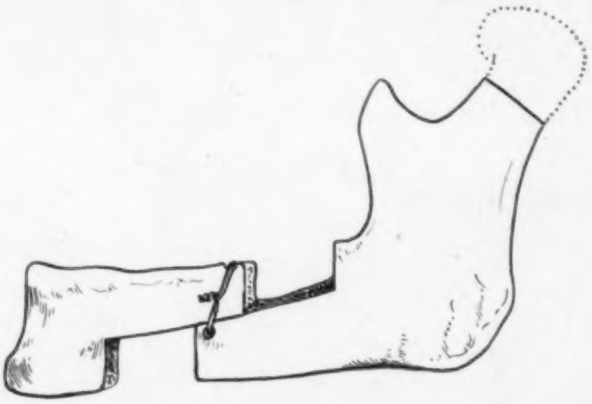

Fig. 218.—Schematic drawing showing sliding osteotomy to be performed on this patient.

Two white acrylic splints had been previously prepared (Fig. 221). The splint for the lower jaw was inserted, and found to fit well. A circumferential wire was inserted at the symphysis (Fig. 222), and twisted over it to hold it in place (Fig. 223). The maxillary splint was inserted next, and steel wire was passed through the previously prepared holes in the beads added at the outer surfaces of both splints. Thus, the two splints were wired together, leaving a feeding space in front (Fig. 224). The change in the outline of the face was

at once noticeable, since the chin was moved to the right into its normal position (Figs. 225 and 226). For further support, a Barton bandage was applied around the head.

Fig. 219.—Drill holes made through mandible to outline the osteotomy.

Fig. 220.—Drill holes are connected to complete osteotomy. Note medicine dropper, *C*, connected with a saline drip for cooling burrs, and suction, *S*, to take up fluid in mouth.

Following the operation, the patient was given an intravenous injection of 1,500 c.c. of 5 per cent dextrose and water, and sulfadiazine therapy was instituted. Two grams of sulfadiazine were given at the start, and 1 Gm. every

six hours for three days; then 1 Gm. each day for three days with sodium bicarbonate.

The postoperative course was smooth, and the patient was discharged on the sixth postoperative day. An x-ray taken at this time (Fig. 227) showed satisfactory position of the jaw. The results of treatment and prognosis were

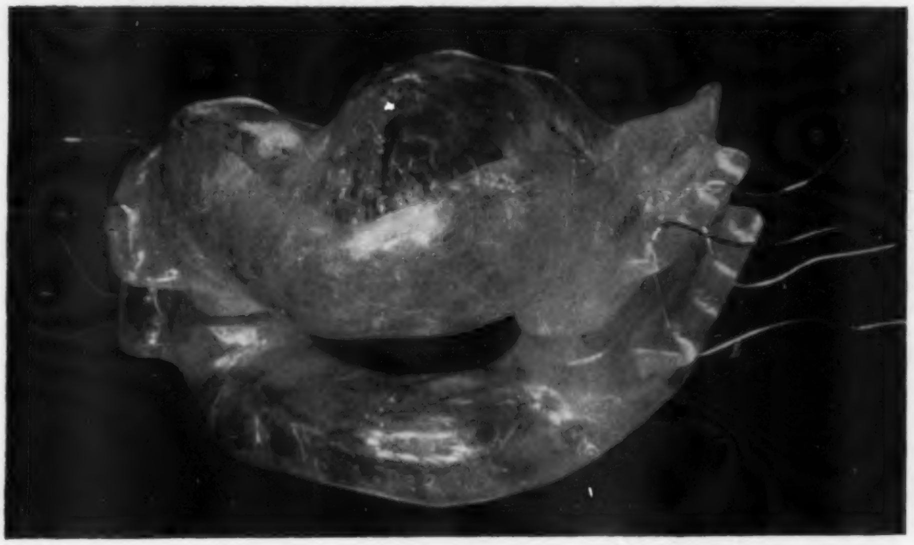

Fig. 221.—White acrylic splints made in two sections. Can be united by wires passed through beads on each side.

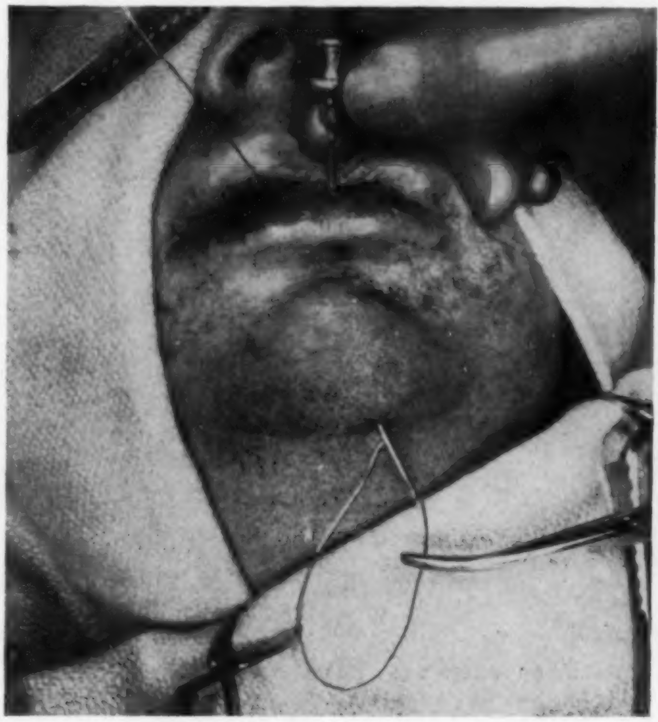

Fig. 222.—Circumferential wire is inserted by means of hypodermic needle to hold mandibular splint in place.

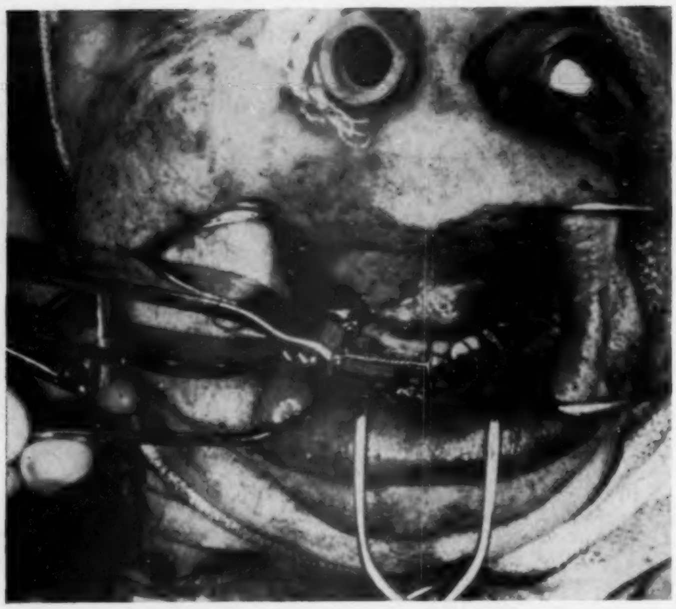

Fig. 223.—Circumferential wire twisted over acrylic mandibular splint.

Fig. 224.—Both splints are inserted and attached by wires to one another on each side.

Fig. 225.—Mandibular deformity before operation.

Fig. 226.—Mandibular deformity corrected by sliding osteotomy.

good. He was seen at the office, where his mouth was sprayed out twice a week until November 22, when the splints were removed because the x-ray showed satisfactory progress in healing. A month later he was advised to see his dentist to have dentures made.

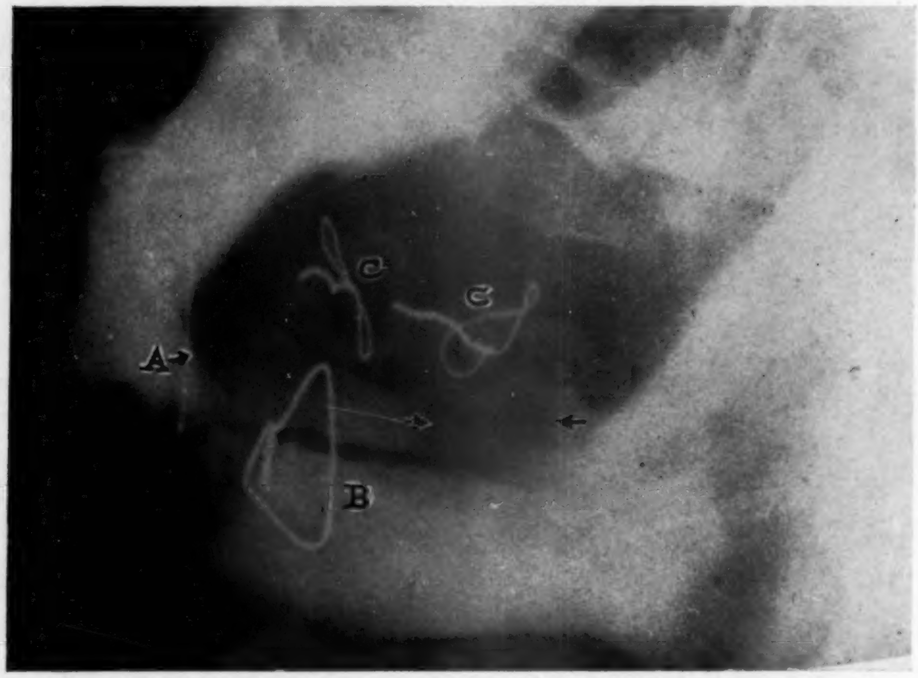

Fig. 227.—Postoperative x-ray showing result of sliding osteotomy. Space gained between arrows measured 1 inch. *A*, Circumferential wire at symphysis; *B*, tantalum wire to hold the two fragments; *C-C*, wires to fasten the two splints together.

Discussion.—The deformity presented by this patient is of interest. It was my opinion that lack of function caused the underdevelopment of the jaw, greater on the side on which the condyle had been injured than on the side in which some motion was possible. Rushton,* however, has a theory that growth of the jaw is governed by the condylar epiphysis, and that injury disturbs epiphyseal growth. The fact that the mental foramen was so far back is evidence that the lack of development took place posterior to this anatomical landmark.

Advancement of the jaw can be accomplished by osteotomy in the vertical ramus or the horizontal ramus. The mandible of this patient was ideal for the latter operation. It offered more positive opportunity for fixation than the operation in the vertical ramus, where the fragments cannot be visibly reduced and fixed by wiring.

*Rushton, M. A.: Growth at the Mandibular Condyle in Relation to Some Deformities, Brit. Dent. J. 76: 57, 1944.

MAY, 1944

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CASE REPORTS FROM THE FILES OF THE REGISTRY OF DENTAL AND ORAL PATHOLOGY

Sponsored by
The American Dental Association

LT. HENRY M. GOLDMAN,* D.C., ARMY OF THE UNITED STATES

THE case reports which are presented in this treatise have been selected from the files of the Registry of Dental and Oral Pathology, sponsored by the American Dental Association at the Army Medical Museum. In each report the diagnosis of the case by means of clinical and microscopic examination is emphasized. Since many of the lesions seen in the oral cavity tend to vary in appearance, it is almost impossible at times to make a diagnosis from the clinical symptoms alone. A biopsy of the tissue in these cases is of extreme importance, in that the morphologic nature of the lesion can be studied. Correlation of both examinations will usually make possible a correct diagnosis.

Care must be employed in choosing the site of biopsy. Tissue from the central portion of a lesion may show marked secondary change so that the histologic picture may be altered. Therefore it is usually best to excise a portion of the adjacent normal tissue with the lesion. Then, comparison between the two may be made; also, the question of invasion can be answered.

The question of histogenesis and pathogenesis are often of considerable importance in determining the nature of the treatment to be instituted. This may be answered in the study of the biopsied tissue. Frequently, a clinical diagnosis must be changed as a result of the microscopic examination, and this naturally influences the treatment. An example of this is malignancy, revealed by biopsy of clinically benign lesions of the lip.

Often a questionable diagnosis may be cleared up by the use of microscopic examination. For example, leucoplakia and lichen planus are common lesions found in the oral cavity. It is important to differentiate between these entities since leucoplakia possesses cancerous tendencies. However, this may occasionally be a difficult procedure from the clinical aspect, in that often one may tend to resemble the other. These two lesions, however, can easily be distinguished histologically, and a positive diagnosis can be made.

FOLLICULAR CYSTS

Follicular cysts are derived from expansion of the follicular sac (enamel organ) of the developing tooth. This disturbance may result in three types of cysts: first, the follicular cyst containing no tooth, this type occurring in the very early stage of tooth development; second, the dentigerous cyst, which forms

*Pathologist and Secretary to the Registry.

after the enamel organ has produced the crown of the tooth; and third, the follicular cyst with odontoma which results from a disturbance of tooth formation as well as production of a cyst.

Since follicular cysts are formed during the developmental stage of the tooth, they are found in young patients. Examination will usually reveal a tooth missing from the arch, but not always, since the follicular cyst may form from a supernumerary tooth bud. The absence of symptoms, however, prevents early discovery unless both a routine dental and radiographic examination are made. Later in life they may become symptomatic.

Dentigerous cysts are easily diagnosed since they are always formed around the crown of a tooth. However, it is always possible that a seemingly dentigerous cyst may, upon microscopic examination, be an ameloblastoma, since the latter tumor has been found to form from the epithelium of dentigerous cysts. Therefore, it is advisable that all cases be studied microscopically. Cahn has shown a transition of odontogenic cysts into ameloblastomas. Thoma and Procter also reported adamantine tissue in the wall of a mandibular cyst. Complete excision of the cyst capsule is advised, therefore, to eliminate a possible adamantinoma.

Fig. 1.—Case 1. Radiograph of dentigerous cyst. (Neg. 77136.)

Case 1

Dentigerous Cyst

(Acc. 99995.) A white male, aged 35 years, complained of tenderness and swelling of the lower left third molar region. Upon intraoral examination, swelling of the retromolar tissue was evident, and no third molar was in position.

Exploration of the area caused marked pain. Radiographs of the left mandible (Fig. 1) showed a large cystic area which extended from the third molar region posteriorly. The third molar had not only been prevented from erupting, but had been forced to move into the ramus with the apex toward the condyle.

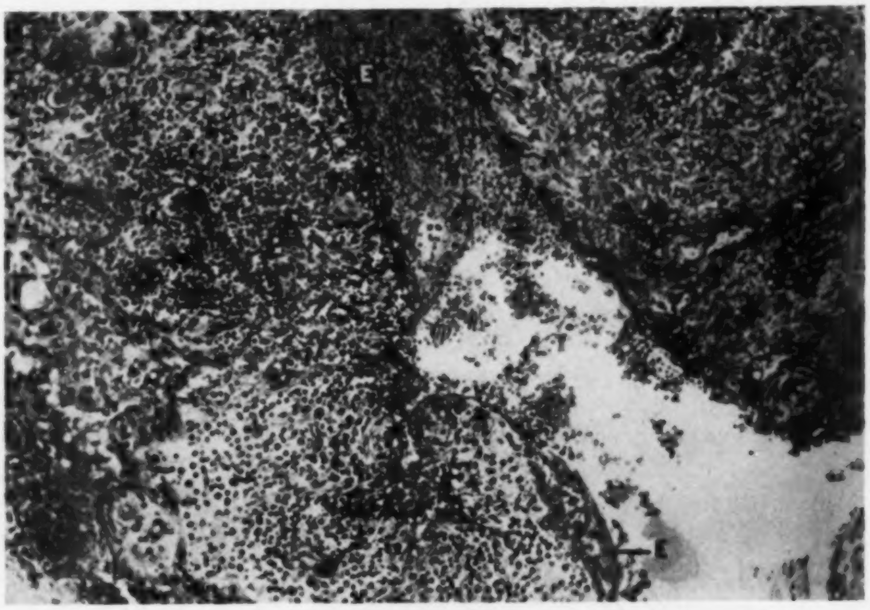

Fig. 2.—Case 1. Photomicrograph showing a marked inflammatory infiltrate in the capsule of dentigerous cyst and the epithelial lining (*E*). (Neg. 77050, ×150.)

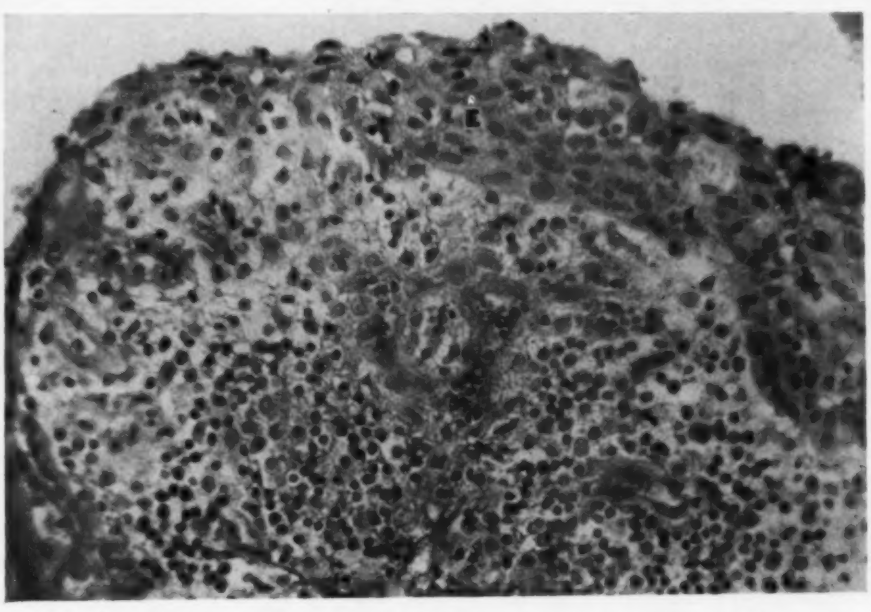

Fig. 3.—Case 1. Photomicrograph showing the epithelial cells lining the cystic space of dentigerous cyst. The inflammatory cells are evident in the epithelium (*E*). (Neg. 77048, ×240.)

There a distinct cortical wall was visible, extending from the neck of the tooth and curving downward, giving the radiographic appearance of a cyst. The superior portion of the cyst had a thin bony encasement.

The microscopic examination of the excised tissue shows a cystic lesion composed of a capsule and a fragmentary epithelial lining (*E*, Fig. 2). The capsule consists of a rather loose but densely infiltrated connective tissue. The inflammatory infiltrate is composed of lymphocytes, plasma cells, histocytes, and polymorphonuclear leucocytes. Numerous blood vessels are also present. The epithelium lining the cystic space is represented only as a few cells in some areas while in others it is of considerable thickness (Fig. 2). Inflammatory cells can be seen in the epithelium (Fig. 3). These cells are present in the cystic cavity as well.

Comment.—This is a typical dentigerous cyst; however, tissue removed at operation should be examined microscopically, for, in many instances, ameloblastomas may be found.

Fig. 4.—Case 2. Radiograph of ameloblastoma. This is the monocystic type. (Neg. 70973.)

AMELOBLASTOMA

The ameloblastoma is a tumor of dental origin arising from either the epithelium of the enamel organ, epithelial rests of the periodontal membrane, mucosal epithelium, or epithelium lining cysts. The tumor may be cystic or solid, or a combination of both. The cystic type is the more common. Some investigators believe that the solid and cystic types represent stages of development rather than distinct types of tumors. The radiographic appearance may be either monocystic or polycystic. The mandible is the usual site, the tumor being rarely found in the maxilla. Cases of these tumors in other bones and in

the hypophysis have been reported. Although ameloblastomas are usually local, a small number which have metastasized have been reported.

Radiographic differentiation of the monocystic type from an odontogenic cyst is difficult and sometimes impossible, and a microscopic examination of the tissue is necessary for diagnosis.

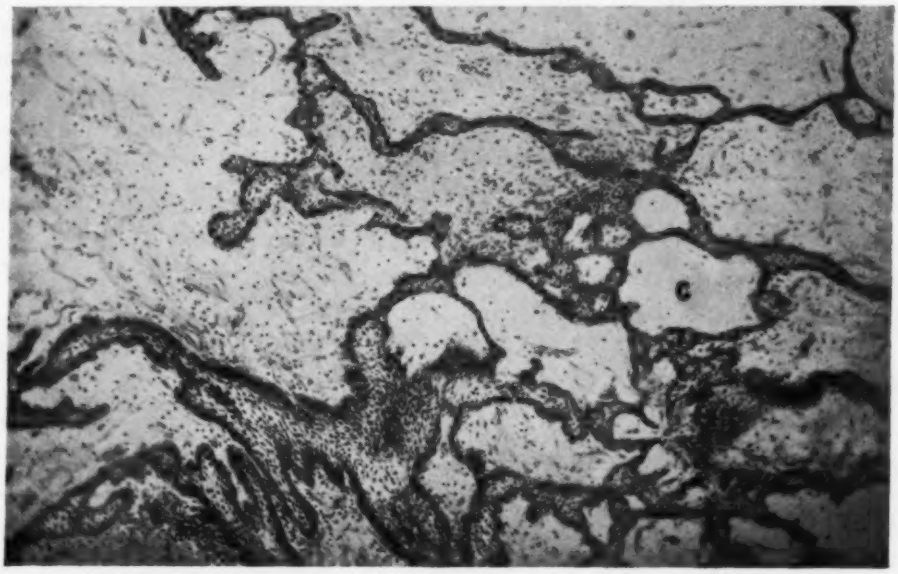

Fig. 5.—Case 2. Photomicrograph of ameloblastoma showing epithelial cells arranged in solid cords and lining cystic spaces (*C*). (Neg. 78090, ×100.)

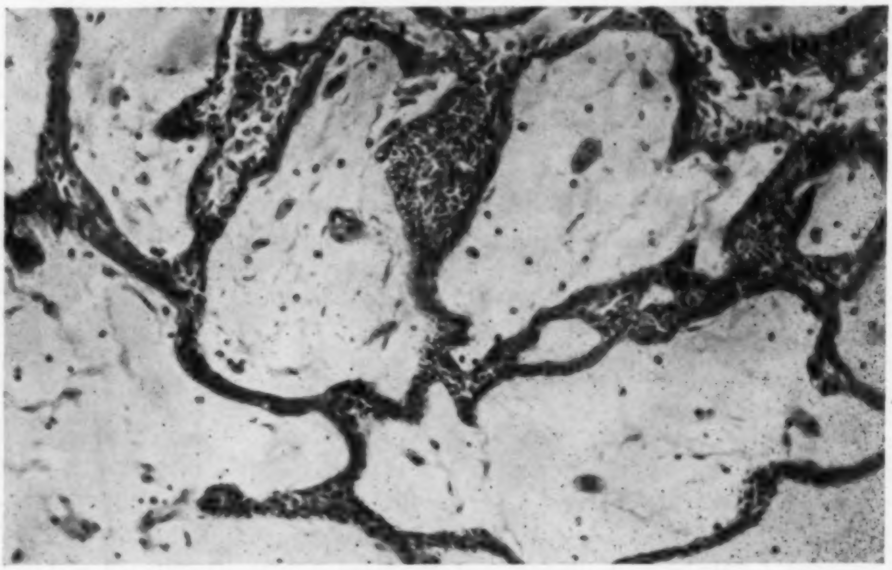

Fig. 6.—Case 2. Photomicrograph of ameloblastoma demonstrating the character of the epithelial cells. (Neg. 78091, ×225.)

Some investigators believe in the conservative treatment of ameloblastomas, if they seem well encapsulated and can easily be enucleated. They believe radical resection is only justifiable as a last resort. Other investigators believe that most of the cases described in the literature show a gradual development,

and that a fairly radical excision performed when the tumor is small may prevent recurrences and repeated serious operations.

Case 2

Ameloblastoma (Cystic Type)

(Acc. 70956.) A Negro female, aged 25 years, presented an acute swelling of the face. Upon intraoral examination, the mouth hygiene was found to be good; the third molar was not in position. On palpation, both the lingual and buccal cortical plates were distended. Radiographic examination revealed a large, smooth-walled, monocystic area the size of an egg, involving the ramus of the mandible. Expansion of the cortex of the anterior portion of the ramus had taken place. In one area there seemed to be a break-through to the outside (Fig. 4). The cystic mass was enucleated and the gross appearance of the specimen was that of an irregular mass of tissue measuring 2 by 1 by 1 cm. The surface was somewhat nodular; the cut surface showed a fairly firm white tissue with minute translucent cysts.

The microscopic examination shows numerous cystic areas surrounded by epithelial cells of the ameloblastic type (*C*, Fig. 5). Some of the cystic areas contain hyaline material with an occasional nuclear fragment, while others are filled with an embryonic appearing myxomatous tissue. In the solid portions of the tumor there are sheets and columns of these cells, some resembling the dental bud. The stroma varies in amount, the purely cystic part having a meager portion while the solid portion contains more abundant stroma. Near the center of the section there is a fibrotic mass which is infiltrated by inflammatory cells. This may be attributed to secondary infection caused by the opening to the oral cavity. Fig. 6 is a high-power photomicrograph illustrating the character of the epithelium and the mucinous change in the connective tissue.

Comment.—Radiographic examination in this case reveals a monocystic lesion which simulates a follicular cyst. Since there is no history of extraction of the third molar and none is present, it may be assumed that this tumor arose either from the epithelium of the tooth germ, or the epithelial lining of a follicular cyst.

Case 3

Ameloblastoma (Solid Type)

(Acc. 61743.) A Negro male, aged 39 years, complained of a swelling and pain of the left side of the face with a discharge into the mouth of four years' duration. The patient first noticed a swelling of the left side of his face, which extended to the temporal region. An incision was made at the left angle of the jaw, freeing a purulent discharge, and the swelling subsided to the present size. Two years ago, he began to have limitation of motion of the mandible. According to the patient, x-rays taken at that time showed an impacted mandibular tooth, but no treatment was instituted.

Examination revealed a fistula in the oral cavity in the left mandibular third molar region, which, upon being probed, exuded a foul smelling, cheeselike material. Marked suppuration was present. Radiographic examination re-

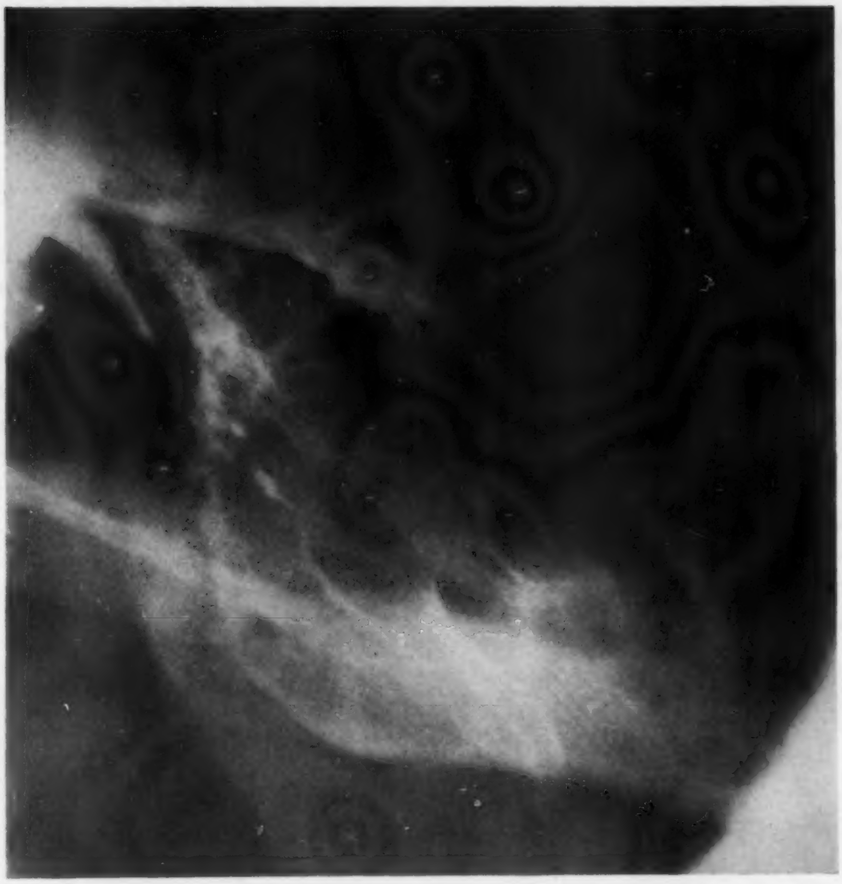

Fig. 7.—Case 3. Radiograph of ameloblastoma. This is the multilocular type. (Neg. 68185.)

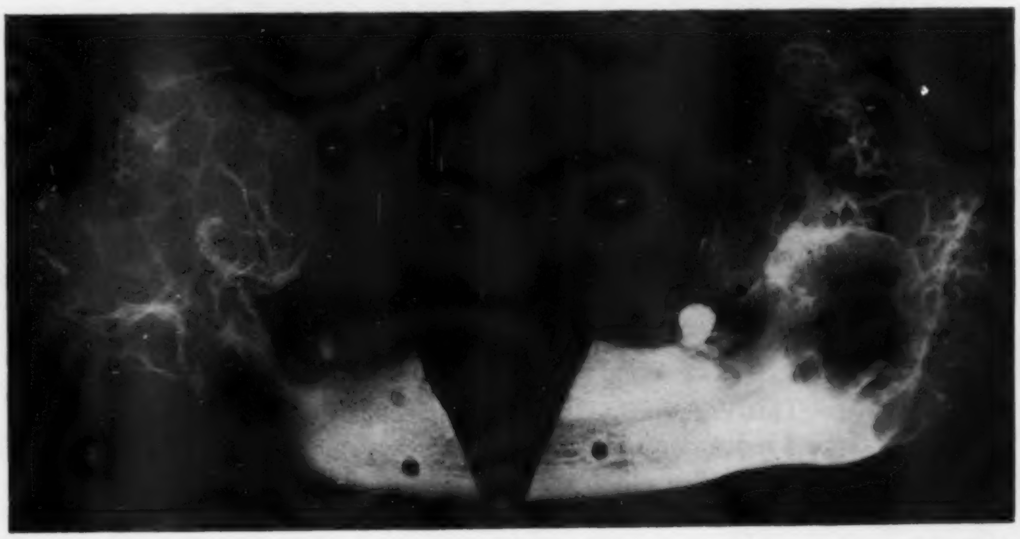

Fig. 8.—Case 3. Radiograph of the resected portion of the mandible of the ameloblastoma. (Neg. 68186.)

vealed a large multilocular lesion about the size of an orange with a molar tooth embedded in the anterior portion of the tumor mass. The lesion extended from the retromolar region to the angle of the jaw and involved the entire ramus (Fig. 7). The radiographic detail is best seen in Fig. 8, which shows the multilocular lesion in the resected portion of the mandible.

At operation, the left ramus and body of the mandible, up to and including the second molar, were removed. Microscopically all sections reveal solid cords, strands, and rests of epithelium growing in a loose fibrous connective tissue resembling the tissue of the dental pulp. The stroma is extremely abundant with comparatively little adamantine tissue. It is composed of a loose reticular-like connective tissue, which in some areas contains numerous cellular elements while in other places it consists of a few stellate cells with long reticular extensions. The nuclei vary in appearance from round to spindle-shaped. A moderate number of blood vessels are present. The epithelial cords resemble the epithelial sprout given off from the dental lamina because of the cellular arrangement and the tendency to form small buds comparable to the earliest stage of enamel organ. Some strands show a tendency to form a stellate reticulum in the central part, the outer layer of cells having a columnar appearance and resembling the ameloblast of forming enamel (Fig. 9).

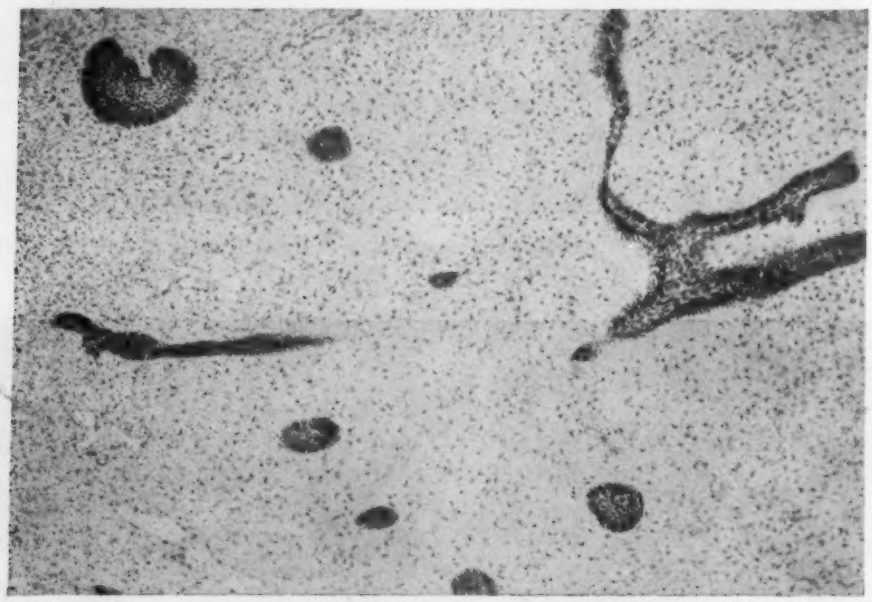

Fig. 9.—Case 3. Photomicrograph of ameloblastoma showing solid cords and nests of epithelial cells in a connective tissue stroma resembling the tissue of the dental papilla. (Neg. 77468, ×100.)

Comment.—This case is of the solid ameloblastoma type, in which the stroma plays the predominant role. In parts its resembles the nondifferentiated connective tissue of the dental papilla; in other areas it has differentiated into a cellular connective tissue not seen in tooth formation. This tumor may be thought to have arisen from both the enamel (ectodermal) and dentine (mesenchymal) organs; therefore, it may be regarded as a mixed tumor of dentigerous origin.

Of interest is the correlation of the radiographic and microscopic appearance of this lesion. From the former, one would expect to find on microscopic examination the cystic type of ameloblastoma. This case may be compared to the cystic type.

Case 4

Ameloblastoma, Arising From a Dentigerous Cyst

(Acc. 102738.) On routine radiographic examination of a white male, a large cyst surrounding the crown of an embedded tooth was found in the lower third molar region. It extended from the second molar area to nearly the posterior border of the ramus and involved the latter almost up to the mandibular notch (Fig. 10). A diagnosis of dentigerous cyst was made. On operation, the cyst contained a purulent material, and brownish granular masses of tissue were attached to the wall.

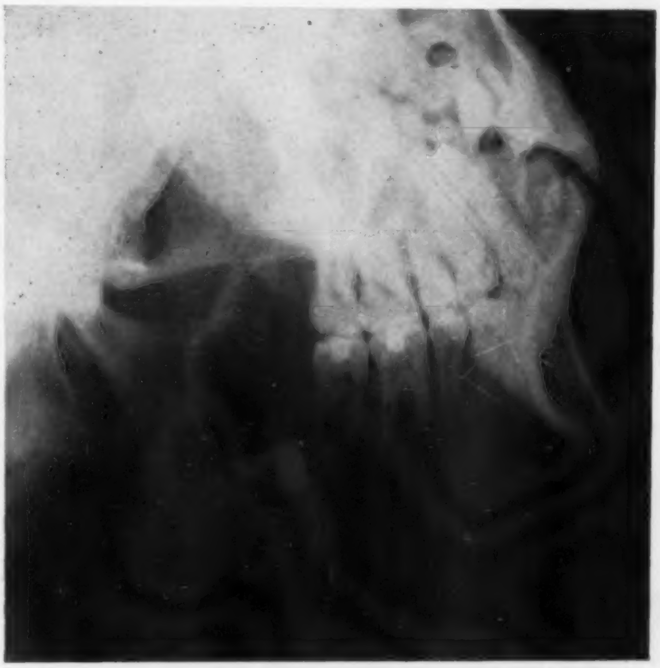

Fig. 10.—Case 4. Radiograph of an ameloblastoma in a seemingly dentigerous cyst. (Neg. 77905.)

Microscopic examination of the tissue reveals an ameloblastoma. The tissue is composed of strands, cords, and large masses of epithelial cells sometimes in alveolar arrangement with cystic formation (Fig. 11). The peripheral epithelial cells contain hyperchromatic oval to spindle-shaped nuclei, and exhibit occasional mitoses (Fig. 12). The cystic cavities are filled with cell detritus or lightly staining, spindle-shaped cells. Very little stroma is evident. In some areas there is evidence of necrosis, and inflammation.

Comment.—This is a very interesting case of ameloblastoma found in what was seemingly a dentigerous cyst. In the files of the Registry, there are several such examples. The etiology of this tumor may be traced to changes in the

epithelial lining of the cystic space. This ameloblastoma was discovered on routine radiographic examination, which once more stresses the importance of this type of service. It also demonstrates the importance of always removing the entire capsule of a cyst since one cannot tell what changes have gone on in the cyst wall.

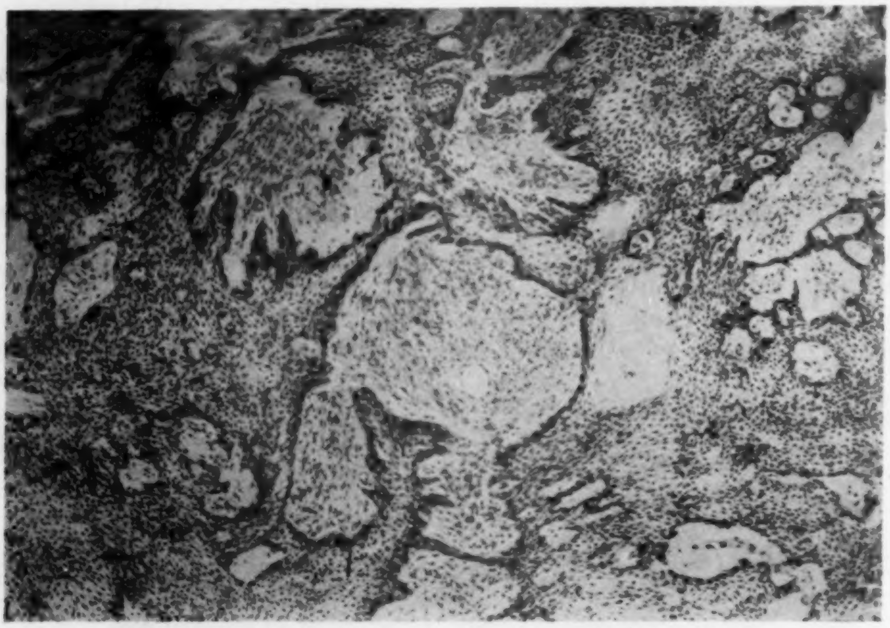

Fig. 11.—Case 4. Photomicrograph of ameloblastoma showing epithelial cells arranged in sheets with peripheral cells resembling ameloblasts. (Neg. 77806, ×100.)

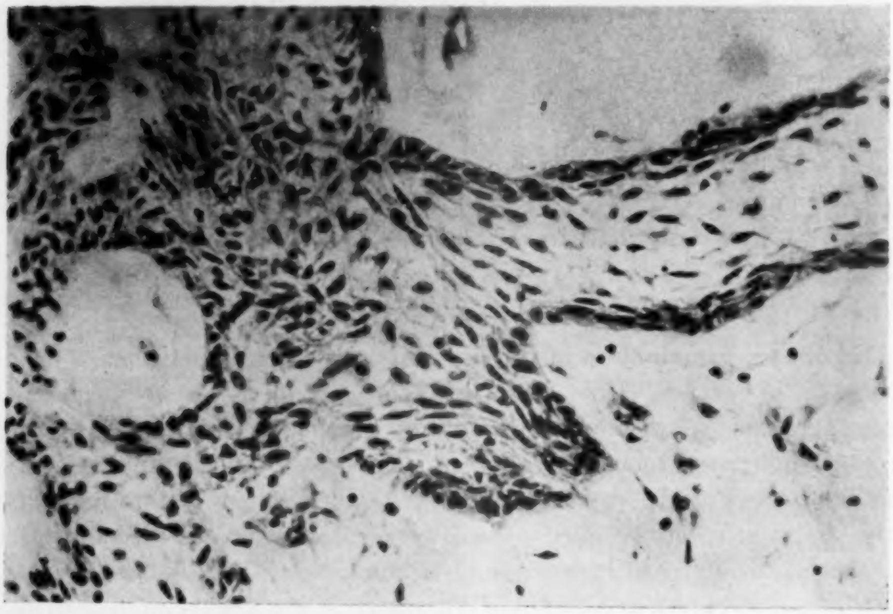

Fig. 12.—Case 4. Photomicrograph of ameloblastoma showing the character of the central and peripheral cells. (Neg. 77807, ×435.)

ODONTOMA

Odontomas are derived from two germ layers: the enamel portion, ectodermal in origin; and the dentine, cementum, and connective tissue portions, of mesenchymal derivation. They are caused by faulty tooth formation. Often they are associated with follicular cysts. Thoma differentiates three types: the geminated composite odontoma in which there is a union of two or more teeth; the compound composite odontoma in which a large number of teeth are found; and the complex composite odontoma in which the tissue is not regular and toothlike, but contains tissue in different stages of tooth development. Sometimes combinations of the three types are found in a single case.

Fig. 13.—Case 5. Radiograph of odontoma. (Neg. 74124.)

Case 5

Complex Composite Odontoma

(Acc. 85173.) A white male, aged 22 years, was admitted to a station hospital complaining of severe pain of one week's duration in the right mandible, with slight pain for three or four weeks prior to that. The past history was essentially negative. The physical examination was negative other than for the finding of an impacted mass in the right mandible. Laboratory examination showed negative urinalysis and Kahn tests. Red blood cell count was 3,950,000, hemoglobin 90 per cent, white blood count 12,400 with 78 per cent polymorpho-

nuclear leucocytes. Radiographic examination showed a rounded mass with a density greater than that of the mandible and completely separated from it by a narrow area of decreased density (Fig. 13). In this mass, an area of still greater density could be distinguished; this resembled somewhat the formation of a crown of a tooth. A diagnosis of a cystic odontoma was made. On the next day of hospitalization the patient developed a severe cellulitis on the right side of the face and was given sulfanilamide and opiates; later, there was a spontaneous eruption of pus noted in the sputum, with relief from pain.

The dental diagnoses at this time were: (1) pericoronitis, acute, suppurative, in region of impacted mass; (2) cellulitis, acute, suppurative, with intraoral drainage in the region of impaction.

Thirteen days later the impacted mass was removed surgically. Healing was uneventful. The gross examination of the specimen showed a rounded, bony-appearing mass weighing 10 Gm. with a thin rough fibrous capsule. The mass varied from 20 to 23 mm. in diameter. Also seen was a molar tooth with its apex angulated at 90 degrees with an attached fibrous mucosa.

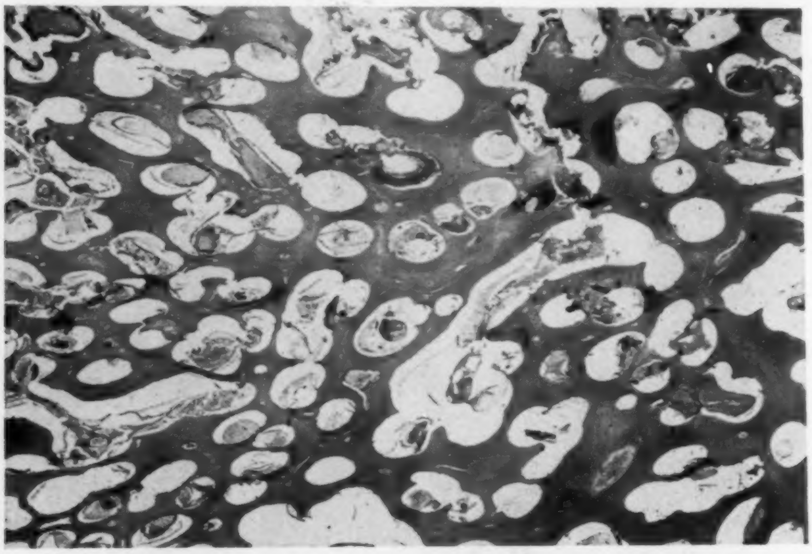

Fig. 14.—Case 5. Photomicrograph of odontoma showing the irregular structural pattern. (Neg. 74094, ×10.)

The microscopic examination shows anastomosing bands of different thicknesses of dentine surrounded by rounded or ovoid spaces, in which an acute inflammatory exudate is seen. The spaces contain organic remnants of enamel matrix in addition to the exudate (Fig. 14). The enamel matrix shows the structure of typical enamel rods. In a few isolated places stratified squamous epithelium is observed, probably derived from the fistula created from the intraoral drainage. Some portions of the dentine contain enclaved cells and give the appearance of osteodentine. The connective tissue in places appears embryonal and may represent the pulps of the aborted teeth.

Comment.—This case may be classified as a complex compound odontoma since a tooth was found as well as irregular dental structure.

TUBERCULOSIS OF THE ORAL CAVITY

Tuberculosis of the oral cavity may be divided into three groups. The first is seen in patients with positive sputum, the infection starting in a superficial wound. The second is found in patients with a general dissemination of the disease, and the third group is found in patients where the spread is by way of the lymphatics. The first is the more prevalent, the latter two being rarely encountered. A shallow superficial ulceration with festooned borders is seen in the first type. The base is usually irregular and is covered by a caseous material. Many cases have been reported in which organisms have migrated through broken-down teeth into the jaw, causing tuberculous osteomyelitis. In the other two groups, there is swelling without the presence of a surface ulceration. However, in the late stages it is sometimes impossible to make the above differentiation, since some of the second and third groups ulcerate.

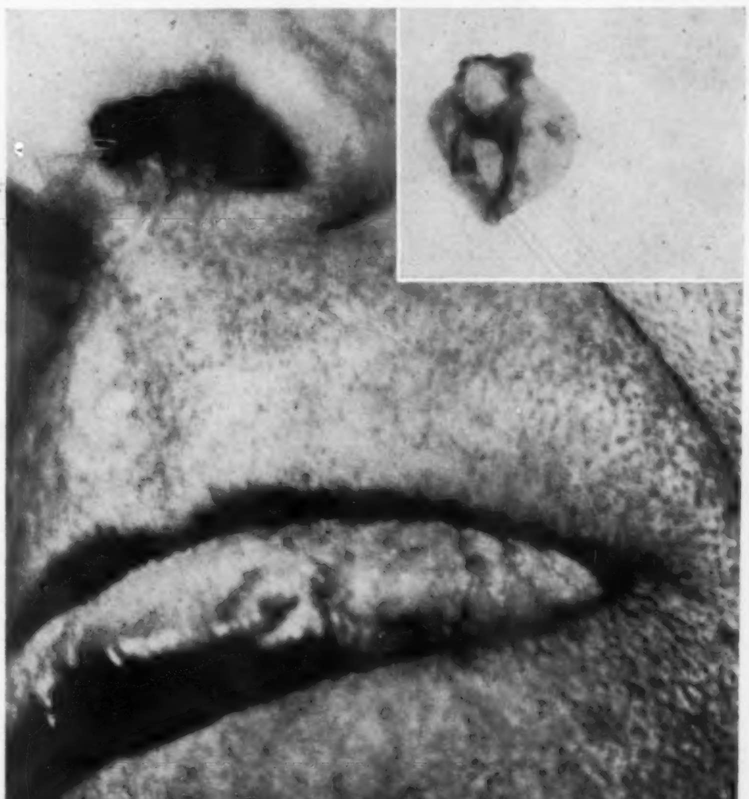

Fig. 15.—Case 6. Tuberculous ulcer of tongue. (Neg. 77742.) Insert shows the lesions after excision. (Neg. 77744.)

Differential diagnosis from other lesions is essential. The distinguishing characteristics are appearance, lymph node involvement, tuberculin reaction, presence of acid-fast bacilli, and animal inoculation. The lesion must be differentiated from carcinoma and syphilis. The former is a hard, nonpainful, indurated lesion with a rolled border; the latter may be differentiated by a

positive Wassermann and presence of *Treponema pallidum*. The tuberculous lesion is usually more shallow and painful.

Tuberculous lesions in the oral cavity may sometimes be found in individuals who are unaware of having a generalized tuberculosis. Since the dentist is frequently the first to see the lesion, he may be instrumental in the discovery of a pulmonary tuberculosis.

Fig. 16.—Case 6. Photomicrograph of ulcer in tuberculosis. *A*, Inflammatory zone; *B*, giant cells. (Neg. 77745, ×25.)

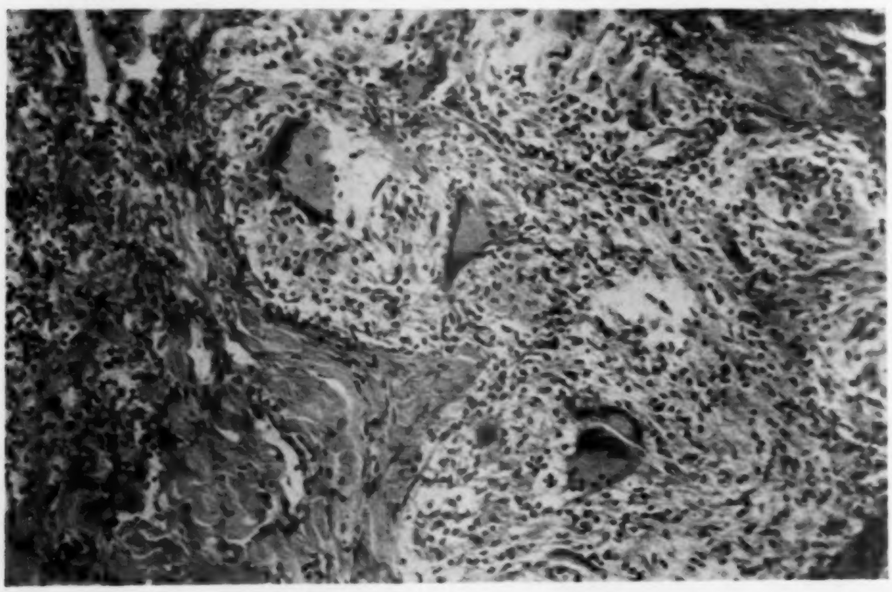

Fig. 17.—Case 6. Photomicrograph showing the giant cells seen in the previous illustration. (Neg. 77743, ×200.)

Case 6

Tuberculous Ulcer of the Tongue

(Acc. 73561.) A retired seaman, aged 48 years, was admitted to the hospital with a past history of three previous admissions for treatment of tuberculous of all lobes of the lungs and a tuberculous enteritis. During the third hospitalization period, he developed a tuberculous ulcer on the mucosal surface of the right cheek. This was excised. The present condition started with a pulmonary hemorrhage following an attack of influenza. Associated with this episode, an ulcer formed on left lateral border of his tongue (Fig. 15). The lesion was deeply furrowed and had two small nodules projecting from the crater of the ulcer. The entire lesion was removed and the gross specimen was an irregular oval piece of tissue 1.5 by 1.1 cm. The surface was convex and covered by a milk-white opaque mucosa.

Microscopic examination reveals a section of tissue covered by a thickened squamous epithelium which has a shallow, irregular ulcer with one island of epithelium remaining in its center (Fig. 16). A polymorphonuclear leucocytic infiltrated membrane covers the ulcer with the base densely infiltrated with the same cells (*A*). A zone of miliary groups of epithelioid cells occasionally with a giant cell in the center can be seen at *B*. Bordering the periphery of the tubercle, a cellular fibrocytic tissue with lymphocytic and eosinophilic cell infiltration is evident. This chronic inflammation involves the underlying submucosa and striated muscle. Fig. 17 shows a high-power magnification of the giant cells seen at *B* in Fig. 16.

Comment.—This case is the second occurrence of an ulcer in the oral cavity of a patient with marked pulmonary tuberculosis.

GIANT-CELL TUMOR

Two types of giant-cell tumors are recognized. The peripheral type found on the gingiva produces a painless swelling which is usually pedunculated, but may have a broad base. It is of firm consistency and the teeth may be separated by the increased growth of the lesion. Often the periosteum is invaded. This is the so-called epulis.

The central form is seen in both jaws and may cause a swelling. The radiographic picture of the giant-cell tumor usually shows a cystic, irregular, lobulated lesion with coarse trabeculations subdividing the defect. It has been observed that the lesion is usually found in parts of cartilaginous origin. Thus, these tumors of the lower jaw are generally found at the symphysis where the os mentis participates in the formation of the mandible. Some are found at the ramus where there is a separate center of cartilaginous ossification.

Some investigators believe these tumors to be true neoplasms, while others feel they are a reactive resorptive lesion. They tend to recur if incompletely excised, but they are not malignant. Geschickter and Copeland believe that giant-cell tumors can be divided into an active progressive osteoclasia and a regressive osteoclasia. In the former, hemorrhage and giant cells predominate; in the latter, cystic bone conditions and fibro-osteosis are predominant. They believe that these tumors are benign and that their growth varies.

Giant-cell tumors must be differentiated from osteoclastomas occurring in generalized osteitis fibrosa of hyperparathyroid origin. Skeleton x-rays as well as blood chemistry should be done. The Institute of Clinical Oral Pathology published a case of a giant-cell tumor in the mandible of a woman, aged forty years, which was excised. It recurred in a different location (ramus) and the patient suffered two pathological fractures in the long bones since the operation on the jaw. A study of the blood chemistry was then advised, which showed the blood calcium to be 11.6 mg. per 100 c.c., the phosphorus 2.86 mg. per 100 c.c. and the phosphatase markedly increased. Radiographic examination revealed cystic lesions in several bones. A diagnosis of generalized osteitis fibrosa of hyperparathyroid origin was made.

Since the peripheral lesion often resembles other epulides of the mouth and the central lesion appears as a cyst, a positive diagnosis can only be made by microscopic examination of the tissue.

Fig. 18.—Case 7. Radiograph of giant-cell tumor. (Neg. 77651.)

Case 7

Central Giant-Cell Tumor of the Mandible

(Acc. 69470.) A white male, aged 27 years, complained of an asymptomatic swelling of the jaw. The past history was irrelevant. The present illness started two months ago when the patient was told by his barber that his chin was swollen. X-rays taken two days later showed a large bone cyst involving the symphysis bilaterally, and extending from the second premolar on one side

to the second premolar on the other. A slight trabeculation of the cystic area was apparent (Fig. 18).

Examination showed an expansion of the labiolingual plates and an infiltration of the lesion into the surrounding soft tissue. A smooth hard enlargement over the mental region on the left side was also found. The anterior cervical lymph nodes were slightly enlarged, round, discrete, and somewhat tender. Laboratory examinations were negative. At operation the lesion was removed in fragments. On sectioning the largest piece, a dark red and white streaked surface was seen.

Microscopic examination reveals a connective tissue which varies from a dense collagenous fibrosis to a loose collection of young fibroblasts. Numerous multinucleated giant cells are scattered through the tissue. No mitotic figures can be found, but abundant blood vessels are evident (Fig. 19).

Comment.—A positive diagnosis in this type of lesion can only be made by microscopic examination of the tissue.

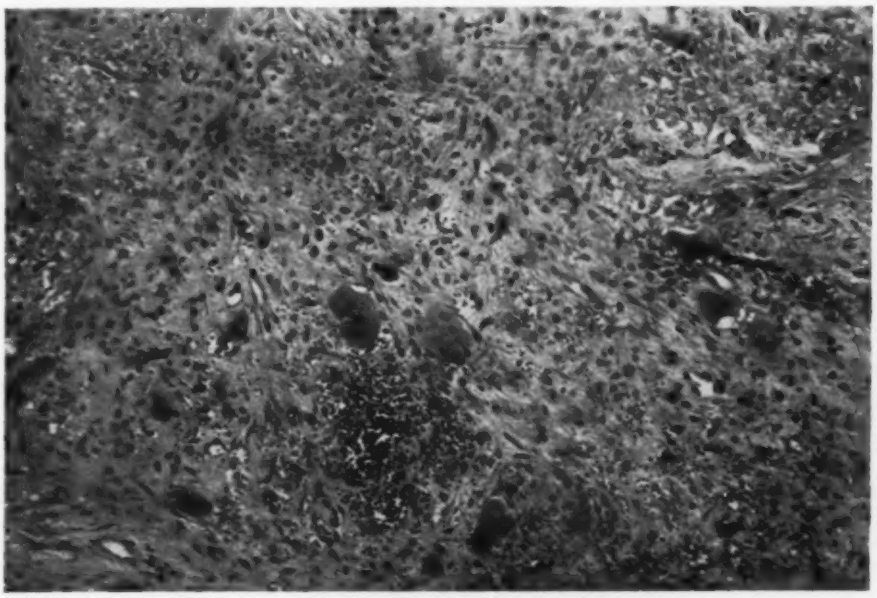

Fig. 19.—Case 7. Photomicrograph of giant-cell tumor, showing the giant cells in a connective tissue stroma. (Neg. 77653, ×175.)

OSSIFYING FIBROMA

This lesion has been termed localized osteitis fibrosa, localized osteodystrophy, hypertrophic localized osteitis, osteofibroma, and fibrous osteoma. The reason for all these names is the various appearances of the lesion; hence no one term seems to fit all of the cases encountered. Most investigators believe that this lesion is one entity and may show various pictures at different stages.

Thoma believes that it is a tumor of medullary origin and as such is a true neoplasm. When there has been an initial replacement of the marrow with fibrous tissue and the formation of new bone trabeculae, he believes that the term fibrous osteoma is applicable. On the other hand, when the fibrous tissue proliferation is more abundant and bone trabeculae are only beginning to form, the term ossifying fibroma is more accurate. Thus, there is an associa-

tion between the two, the ossifying fibroma apparently representing the initial neoplastic response.

Furedi groups these tumors into two types, the hyperostotic and hypostotic. The former is described as a condition where the marrow is displaced by a cellular fibrous tissue in which are deposited newly formed trabeculae. Simultaneously there is evidence of bone resorption but to a much lesser degree. In the hypostotic type the marrow spaces are widened, the bony trabeculae undergoing resorption. New bone formation is absent. The tissue is made up of a loose connective tissue in which are seen many blood vessels.

The lesion is said to be more frequent in females and is usuallly seen in the second and third decades of life, although it may occur at any age. The usual symptom is asymmetry of the face or a disturbance of occlusion. Some investigators believe the onset is associated with trauma. It is slow-growing and nonpainful. A differential diagnosis is made on the basis of radiographs, duration, course, and biopsy.

Fig. 20.—Case 8. Radiograph of ossifying fibroma. (Neg. 77749.)

Case 8

Ossifying Fibroma of the Mandible

(Acc. 91556.) A white male, aged 22 years, had had a bad toothache seven years previously. The tooth was extracted after a long time had elapsed. Difficulty was experienced and the tooth was broken during its removal, but healing

was uneventful. About a year later, the patient noticed a swelling on the right side of the mandible, which gradually increased to its present size.

Radiographic examination revealed a large mottled rarified area extending from the first premolar to the second molar and to the lower border of the mandible distending the cortical plate downward (Fig. 20). Examination revealed no numbness of the lip even though on radiographic appearance the mandibular canal seemed obliterated. At operation, the tissue from this area was removed, and the specimen consisted of several pieces, the largest measuring 3 by 1.5 cm. There was some grittiness on cutting the tissue and the surface was white.

The microscopic examination of the largest piece of tissue reveals a section of connective tissue (Fig. 21) in which are seen spicules of a normal-appearing bone (*A*) and very many areas of focal calcification (*B*). The connective tissue is fairly dense with numerous blood vessels and in one area fibrous bone is being formed.

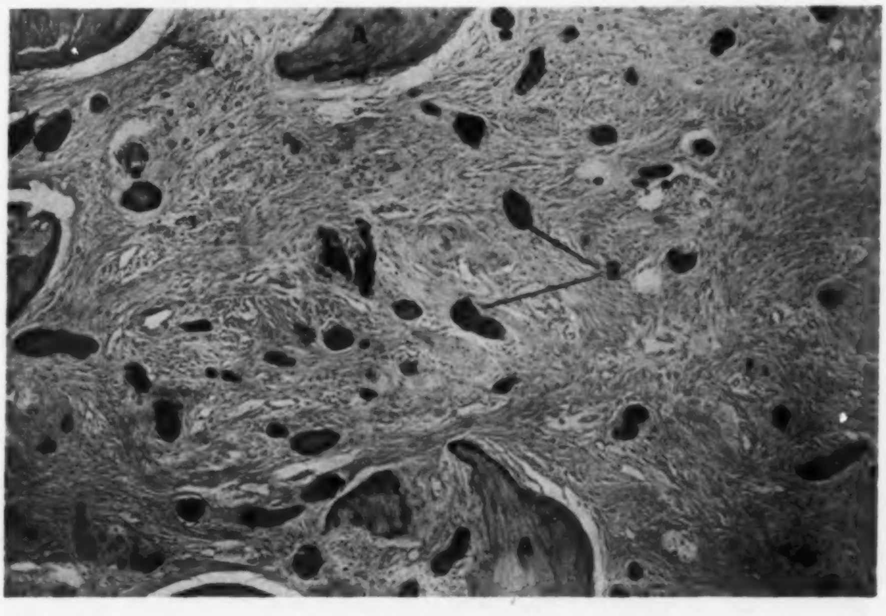

Fig. 21.—Case 8. Photomicrograph of ossifying fibroma, showing areas of focal calcification (*B*) and mature bone (*A*) in a connective tissue stroma. (Neg. 77748, ×100.)

Case 9

Ossifying Fibroma of the Maxilla

(Acc. 94992.) A white male, aged 23 years, noticed, one year previously, a pea-sized swelling on the left upper jaw at the first molar region. It grew rapidly into an ovoid mass on the palate and apparently extended into the antrum, for the latter could not be filled with lipiodol. No lymph adenopathy was present. Radiographic examination revealed a cystic rarefaction between the first molar and second premolar which extended deeply into the alveolar bone (Fig. 22). Extention into the sinus could not be determined. At operation, the tumor extended from the second premolar to and including the third molar and up to the malar process through the floor of the antrum which was full of polypi.

Fig. 22.—Case 9. Radiograph of ossifying fibroma. (Neg. 77005.)

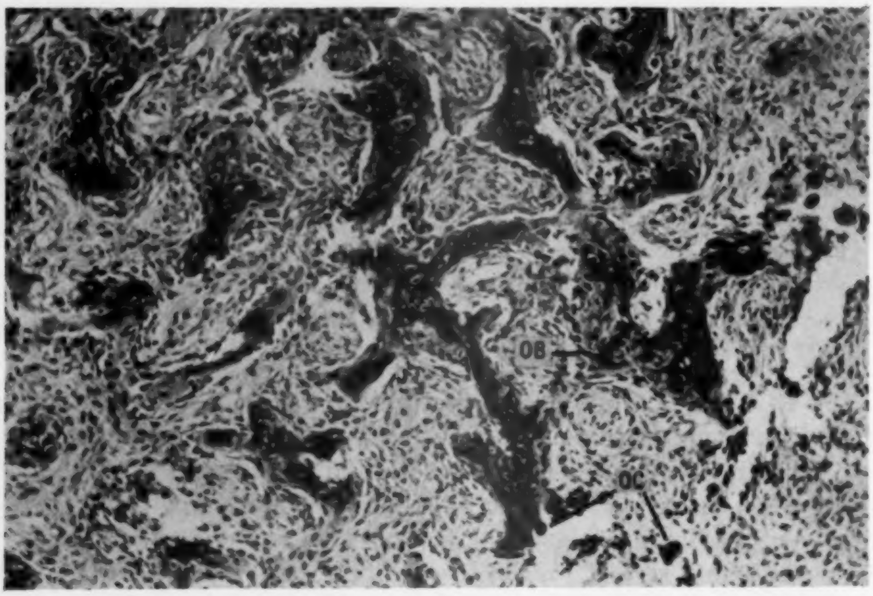

Fig. 23.—Case 9. Photomicrograph of ossifying fibroma, showing immature bone trabeculae in a connective tissue stroma. Osteoblastic (*OB*) and osteoclastic (*OC*) activity are evident. (Neg. 77917, ×165.)

The microscopic examination reveals a tissue consisting of bony spicules, many of them with prominent osteoblastic rims, all embedded in a loose fibroblastic matrix (Fig. 23). For the most part the bony spicules seem immature, with large haversian canals with fairly large bone cells. Most trabeculae are encased by a rim of osteoid tissue. Osteoblasts are seen at *OB*, osteoclasts at *OC*. The arrangement of the trabeculae is haphazard with no definite architecture. The loose fibrous marrow contains abundant round fibroblasts. Under high power the character of the bone formation can be seen (Fig. 24).

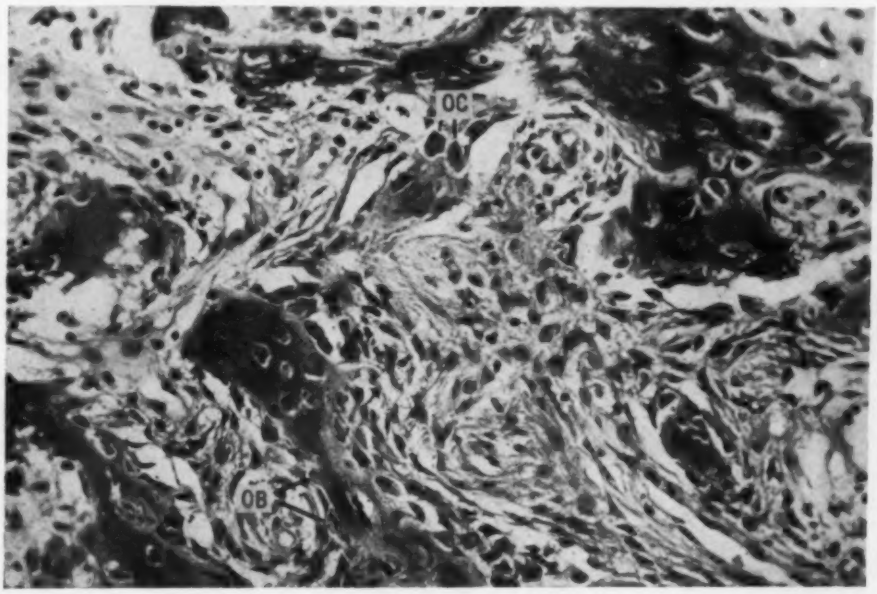

Fig. 24.—Case 9. Photomicrograph of ossifying fibroma showing marked osteoblastic activity (*OB*). Osteoclasts can be seen at *OC*. (Neg. 77918, ×350.)

Comment.—Cases 8 and 9 show two types of lesions classified under the general heading of ossifying fibroma. In Case 8, there are areas of focal calcification present in a fibrous narrow. Adult bony trabeculae are seen which show evidence of a past resorptive phase, but are at present inactive. On the other hand, in Case 9, the entire tissue seems active, with no old bone evident. Exact terminology according to Thoma would title Case 8 an ossifying fibroma, while Case 9 would be called a fibro-osteoma.

Case 10

Ossifying Fibroma of the Mandible

(Acc. 93943.) The past history of a white female, aged 40 years, revealed a surgical removal of what was thought to be a radicular cyst which extended from the lower right second premolar to the second molar. On pathologic examination of the excised tissue, it was impossible to confirm this diagnosis. The tissue showed definite portions of a cyst wall, but no epithelial lining was visible. Its outer border was fairly sharply demarcated from the surrounding structure. The wall proper presented a background of proliferating fibroblasts, some areas being extremely cellular to a degree suggesting neoplasia. An ir-

regular scattering of giant cells of the osteoclastic type and many bone trabeculae were present, some apparently proliferating, others degenerating; zones of osteoid tissue were noted (Fig. 25).

The patient was placed under observation, and in a short time recurrence of the mass was evident. External enlargement could be demonstrated. On digital examination a softening with fluctuation could be felt through the previously operated area in the buccal alveolar ridge, and also on the lingual surface of the mandible. Figs. 26 and 27 show the radiographs of the lesion at this time. A marked resorptive process is evident, which extends to the lower border of the mandible and has broken through the periosteum to the outside. No radiographic evidence of a sclerosing process can be seen. It was deemed advisable to resect that portion of the mandible.

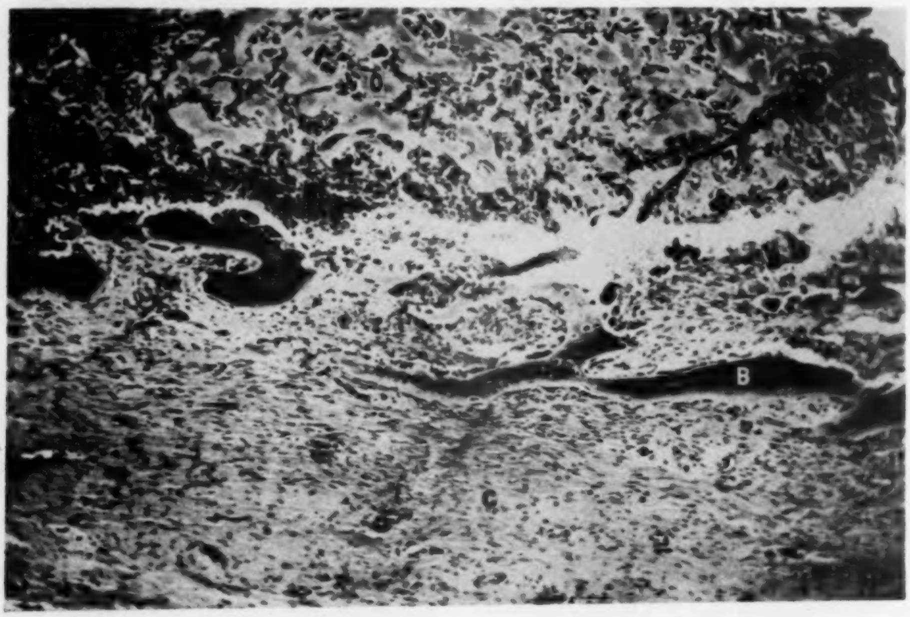

Fig. 25.—Case 10. Photomicrograph of ossifying fibroma. *C*, Connective tissue; *O*, osteoid; *B*, bone. Radiograph appeared as a radicular cyst. (Neg. 77572, ×100.)

Microscopic study shows the tissue to be covered by a thickened stratified squamous epithelium, beneath which there is an infiltration of plasma and round cells. Some of the tissue, apparently taken deeper in the lesion, shows irregular islands and areas of a homogeneous, pink-staining matrix, which are surrounded by a layer of medium-sized, fairly deeply staining cells having round or oval, deeply staining nuclei (Fig. 28). In some of these areas there were spicules showing calcium deposition (Fig. 29). The diagnosis of ossifying fibroma of the fibro-osteoid-osteoma type was made.

Comment.—Comparison of the radiograph of this case and those of the typical cases of ossifying fibroma is of interest. In most of the latter, there is no distinct demarcation of the process. The cortex is thinned out and there is no periosteal bone formation. A typical stippling of the spongiosa is usually seen. The sclerosing type is radiopaque. In the present case, there is marked

expansion of the mandible with a marked osteolytic appearance. No stippling and demarcation can be seen.

Comparison between the radiographic and microscopic appearance of this lesion is of interest in that while there is a diffuse production of bone in the

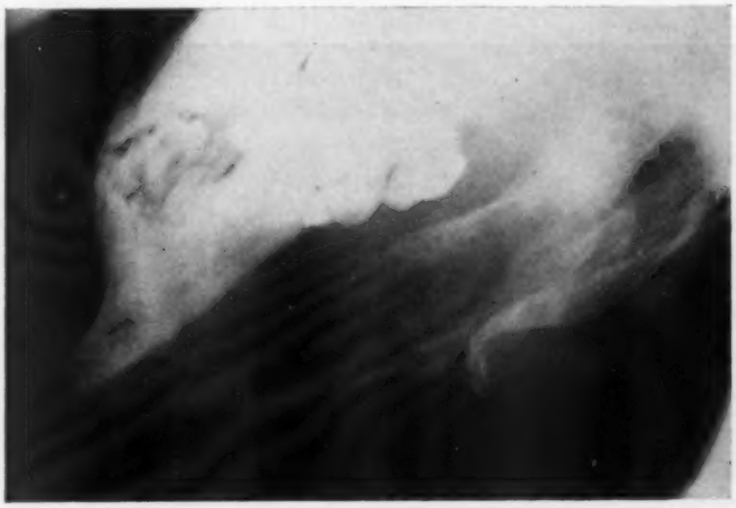

Fig. 26.—Case 10. Radiograph of ossifying fibroma before resection. Lateral view. (Neg. (Neg. 77627.)

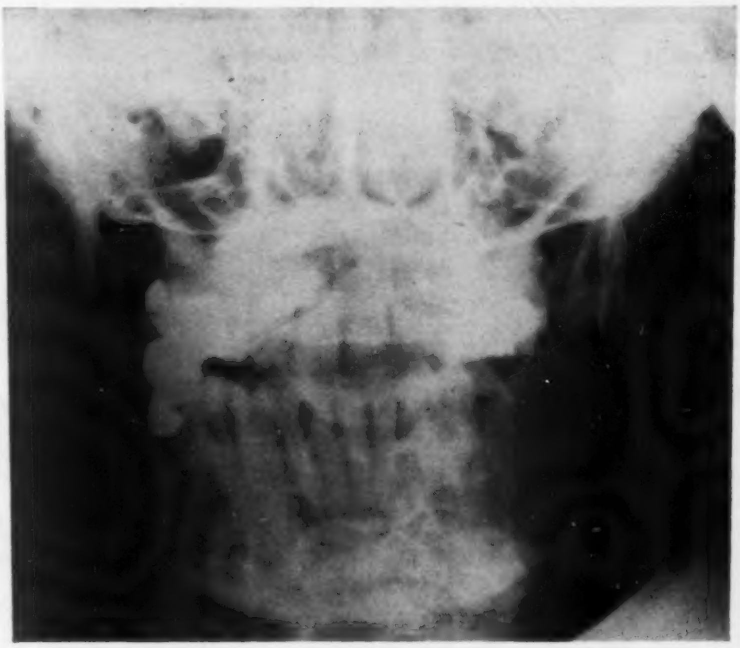

Fig. 27.—Case 10. Radiograph of ossifying fibroma before resection. Anterior-posterior view. (Neg. 77741.)

lesion, none is evident in the x-ray. Thus, it may be concluded that the new tissue is ''osteoid'' rather than true calcified bone.

The tumor soon recurred and its growth was far faster than usual in ossifying fibroma. The latter is usually a slow-growing tumor and treatment is

usually conservative. However, in this case the radiographic appearance of the lesion and its rapid course justified the resection of the mandible. This lesion may be considered as the type which remains poorly calcified and is termed fibro-osteoid-osteoma. It must be differentiated from an osteogenic sarcoma. Comparison with Cases 9 and 10 should be made.

Fig. 28.—Case 10. Photomicrograph of ossifying fibroma showing marked osteoid formation (*B*) in a connective tissue stroma. An inflammatory infiltrate is present at *A*. (Neg. 77571, ×10.)

Fig. 29.—Case 10. Photomicrograph of ossifying fibroma showing calcification of some of the osteoid tissue. (Neg. 77573, ×100.)

NEUROFIBROMA

The neurofibroma is generally benign, composed of connective tissue the exact origin of which is still a matter of dispute. Some pathologists attempt to differentiate tumors arising from the perineural connective tissue from those arising from the Schwann cells of the neurilemma. The differentiation is still not definitely settled.

In some cases this tumor occurs in multiple form and is called neurofibromatosis or von Recklinghausen's disease. In this clinical entity there are multiple tumors of the skin, possibly also of the peripheral and cranial nerves, pigmentations of the skin, and endocrine disturbances. It is well, therefore, to eliminate this disease by a general examination when a neurofibroma is found.

Fig. 30.—Case 11. Radiograph of central neurofibroma. The lesion follows the mandibular nerve and does not seem related to the teeth. (Neg. 76248.)

Case 11

Neurofibroma of the Mandible

(Acc. 97380.) A white male, aged 33 years, had recurrent pain after the filling of a cavity of a mandibular tooth. A radiograph revealed a large cystic lesion that extended from the lower first premolar region to the center of the ramus of the mandible (Fig. 30). The lesion was diagnosed as a radicular cyst. Laboratory tests were all negative.

The five teeth on that side were extracted and the lesion was removed through a window made in the buccal plate of the body of the mandible from the second premolar to the second molar. Two large pieces of soft, friable, smooth tissue were removed. Sectioning of the tissue revealed a soft, homogeneous, yellowish-brown surface containing minute brown foci.

The microscopic examination shows a loose reticulated type of mesenchymal tissue containing occasional nerve filaments. A fragment of a myelinated nerve is noted within reticulated tissue (*N*, Fig. 31). The cells of the tumor are occasionally arranged in bundles and in whorls. The nuclei are small and either

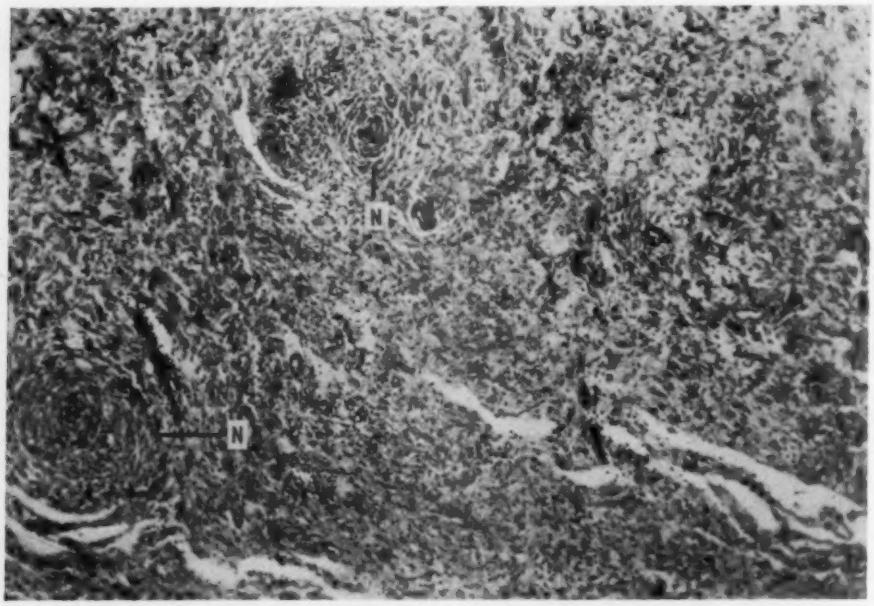

Fig. 31.—Case 11. Photomicrograph of neurofibroma showing a characteristic structural pattern. *N*, Medulated nerve. (Neg. 77378, ×125.)

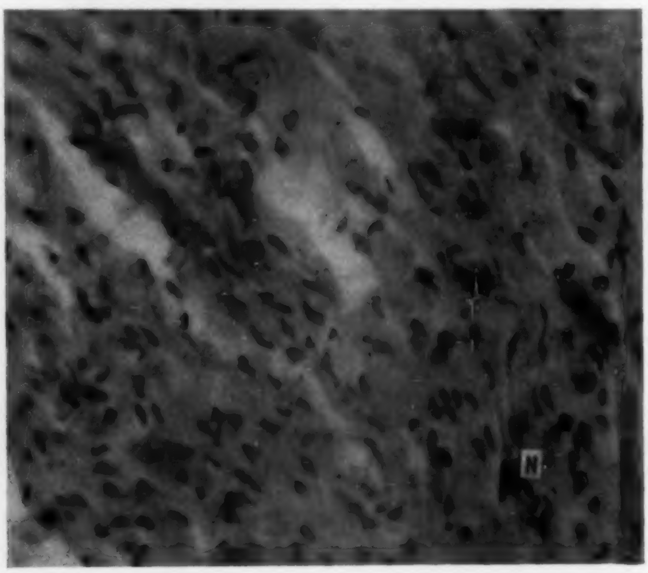

Fig. 32.—Case 11. Photomicrograph showing the cellular structure in neurofibroma. Palisading of nuclei is evident at *N*. (Neg. 77377, ×550.)

round, oval or spindle-shaped. No mitotic figures are seen. The cytoplasm is scanty and occasionally arranged in pointed streamers at each pole of the spindle-shaped nuclei. Infrequent bundles of hyalinized collagen and a few small blood vessels are present. The high-power photomicrograph (Fig. 32)

shows the nature of the cells and their palisaded arrangement (*N*), a characteristic feature of the tumor.

Comment.—The radiograph discloses that this cystlike lesion in the mandible follows the course of the mandibular nerve, and that the left mental foramen is greatly enlarged. These radiographic findings support the diagnosis of a neurofibroma involving the mandibular nerve.

Zilkens reported a similar case which occurred in a 17-year-old boy. He had had a swelling of the chin for eleven years. Intraorally it presented a swelling the size of a pigeon's egg covered by a normal mucosa. No permanent teeth were present. Therefore, a follicular cyst was suspected and seemed to be confirmed by radiograph although the picture was not typical. The tumor was excised and found to consist of glossy transparent masses which on microscopic examination proved to be a neurofibroma. Healing was prompt and uneventful.

Fig. 33.—Case 12. Appearance of patient with neurofibroma of antrum. (Neg. 77430.)

Case 12

Neurofibroma of Antrum

(Acc. 96438.) A white male, aged 28 years, complained of a painless swelling of the right side of his face which was of about two months' duration. The swelling was first noticed after the extraction of a tooth of the right upper jaw. He had had nasal obstruction on the right side since that time. Examination

Fig. 34.

Fig. 35.

Fig. 34.—Case 12. Radiograph of neurofibroma (A. P. view) showing cloudy sinus and destruction of the walls. (Neg. 77433.)

Fig. 35.—Case 12. Radiograph of neurofibroma (A. P. view) showing the extent of the lesion into the petrous portion of the temporal bone. (Neg. 77432.)

was negative except for a swelling of the right side of the face. X-rays showed a cloudy right maxillary sinus with destruction of the walls. X-rays of the lungs and skeleton were negative for evidence of other tumors. A Caldwell-Luc operation was performed and a piece of tissue was removed for biopsy. A diagnosis of neurofibroma was made.

The patient was transferred to another hospital for further treatment and disposition. Examination upon arrival showed this man to be in perfect health except for a painless swelling over the right cheek (Fig. 33). This swelling was hard and about twice the size of the normal left cheek; no lesions were found in the nose although the patient thought that he could not breathe well on the right.

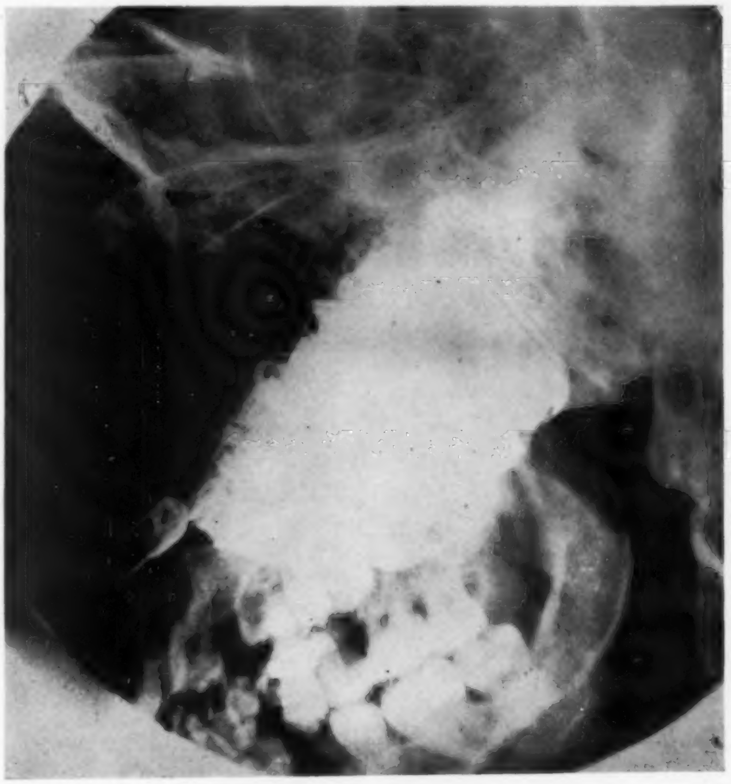

Fig. 36.—Case 12. Radiograph, postoperative, of neurofibroma, showing the extent of the tumor. (Neg. 77435.)

Radiographs showed destruction of the lateral wall of the right maxillary sinus, destruction of the right zygomatic arch, and thinning of the infraorbital margin (Figs. 34 and 35). A deformity of the petrous portion of the right temporal bone was also seen.

The anterior and lateral walls of the maxillary sinus were removed by a modified Caldwell-Luc operation and the entire sinus was found filled with a yellowish lobulated tumor mass. This tissue was removed with curette and forceps, leaving defects in the floor of the orbit and of the antrum. Pulling on the tumor caused the eye to move. The tumor had also grown through the posterior wall of the antrum and into the zygomatic region. To control the pro-

fuse hemorrhage, the external carotid artery was ligated. The antrum was packed with bismuth iodoform gauze. A nasoantral window was made through the right inferior meatus and the gauze strip was brought through this window.

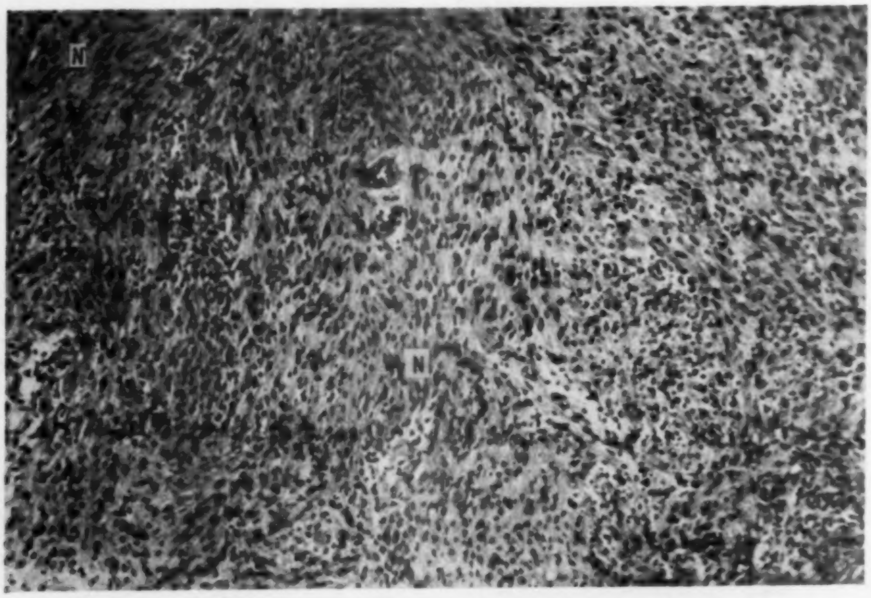

Fig. 37.—Case 12. Photomicrograph of neurofibroma showing palisading of cells (*N*). (Neg. 77426, ×235.)

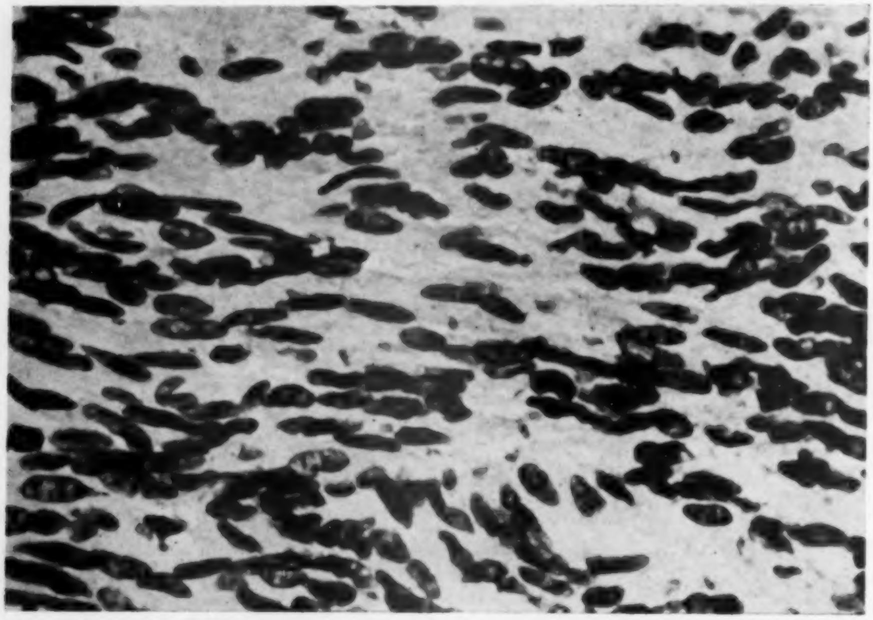

Fig. 38.—Case 12. Photomicrograph of neurofibroma showing spindle-shaped, wavy nuclei. (Neg. 77427, ×765.)

The patient had been given two pints of whole blood and one pint of plasma during the operation and was in fair condition on leaving the operating room. A postoperative x-ray is seen in Fig. 36.

Microscopic examination of the tissue shows masses of spindle-shaped cells arranged in long bundles which interlace irregularly (Fig. 37). The spindle-shaped cells show a variable amount of intercellular material and in some areas there is a definite suggestion of palisading (*N*). In other areas, the tissue appears relatively loose and somewhat myxomatous with areas of degeneration. Under high power (Fig. 38), the wavy nuclei of the spindle cells can be seen clearly.

The patient made an uneventful recovery. Before discharge from the hospital a careful examination was done by the Neurosurgical Service. No signs or symptoms of any cranial nerve involvement were evident except for analgesia of the second division of the right fifth nerve which was probably due to the cutting of this nerve at the time of operation. Even though there was radiographic evidence of intracranial involvement of the right petrous bone, there were no signs or symptoms evident.

Comment.—The symptoms of this case started with the extraction of a tooth. It is probable that the tumor was present but started to proliferate afer the extraction.

Fig. 39.

Fig. 40.

Fig. 39.—Case 13. Radiograph of "myxoma." The lesion has the appearance of a dentigerous cyst. (Neg. 76788.)

Fig. 40.—Case 13. Radiograph, postoperative, of "myxoma." (Neg. 76789.)

MYXOMA

Myxoma in the pure form is a rare tumor, but does occur as a fibromyxoma or chondromyxoma. The term "myxoma" applies to a gelantinoid type of tissue with loose branching fibrillar stellate cells. In most instances, the characteristics are the result of edema in an ordinary fibroma. In the oral cavity they usually occur on the gingiva and in the palate. The records of the majority of cases mention intense, persistent pain as a chief complaint. A constant radiographic

finding is the somewhat mottled appearance of the lesion with an intact bony shell.

Attention has been drawn to the danger of local recurrence of these tumors in spite of their benign appearance and even though they are entirely encapsulated and easily enucleated. It has also been noted that sarcoma may develop in a benign tumor containing myxomatous tissue.

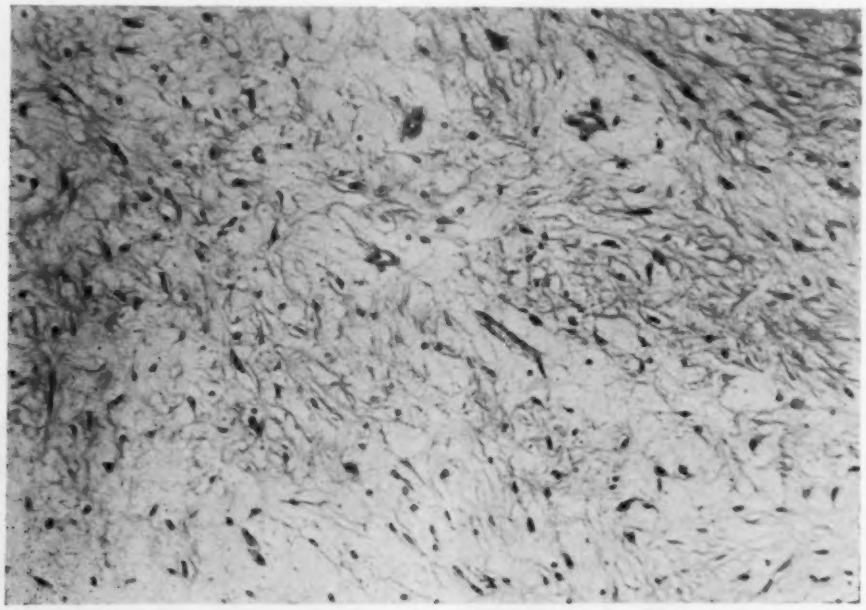

Fig. 41.—Case 13. Photomicrograph of "myxoma" showing the myxomatous structure of the tumor. (Neg. 77747, ×200.)

Case 13

"Myxoma" of the Maxilla

(Acc. 98475.) A white male, aged 23 years, complained of pain of the right upper jaw. Clinical examination revealed a deciduous canine in place and a large swelling of the buccal plate of the maxilla from the lateral incisor to the second molar. The maxillary bone had the consistency of an eggshell. The radiograph showed a large cystic area into which an impacted permanent canine extruded (Fig. 39). At operation, the entire mass was easily enucleated. The postoperative radiograph is seen in Fig. 40. The tissue was white and of soft cartilaginous consistency; on section, the surface had a similar appearance to the external surface. Thin, tenacious, colorless material exuded from the tissue.

Microscopic examination reveals a tissue composed of diffusely scattered, small, fusiform and stellate cells with many delicate radiating, anastomosing and interlacing fibrillary processes (Fig. 41). The intercellular substance is basophilic mucoid material including a few small round vacuoles and showing slight lymphocytic infiltration around the few blood vessels. The architecture is the same in all areas, and no mitotic figures are evident.

Comment.—A similar case to the one presented was published by the New York Institute of Clinical Oral Pathology. A 25-year-old woman complained of a dull pain in the region of the right maxillary first molar. Examination re-

vealed a marked swelling. On x-ray examination a honeycombed osteolytic change was noticed which could not be positively identified. At operation the lesion was found to be made up of a yellowish-white tissue which on microscopic examination revealed the characteristics of a pure myxoma. The radiographs of this case and of the one reported above are identical.

An interesting feature in this case is that the tumor was associated with an impacted cuspid. On the basis of clinical and radiographic evidence alone, it is entirely probable that a diagnosis of dentigerous cyst would be made. Only by the microscopic examination is it possible to learn the true nature of the lesion.

Fig. 42.—Case 14. Radiograph of mixed tumor showing cystic lesion.

MIXED TUMORS

The mixed tumors of the salivary gland type may occur in the oral region in the salivary glands, in the lips, palate, tongue, buccal mucosa, and also rarely as a central jaw tumor when it may be encapsulated and localized. It is a benign lesion, but it may recur after incomplete excision, and may become locally invasive. They vary in rapidity of growth. McFarland (1936) reviewed 300 mixed tumors of which 69 had recurred, and concludes that it is impossible to relate the histopathology to the prognosis.

There are two principal views of the origin of these tumors. The first of these is that mixed tumors are embryonic tumors of local origin being derived

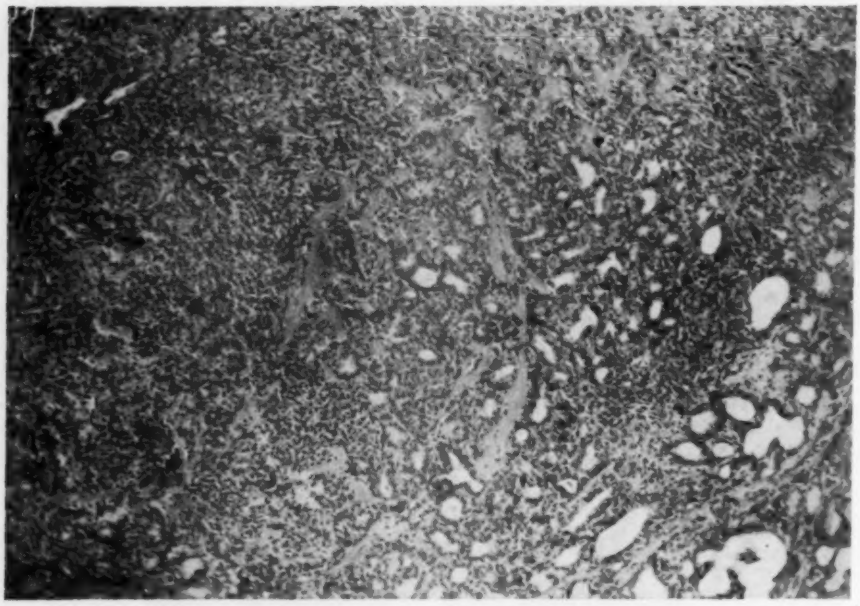

Fig. 43.—Case 14. Photomicrograph of mixed tumor showing a general pattern characteristic of this tumor. (Neg. 77140, ×100.)

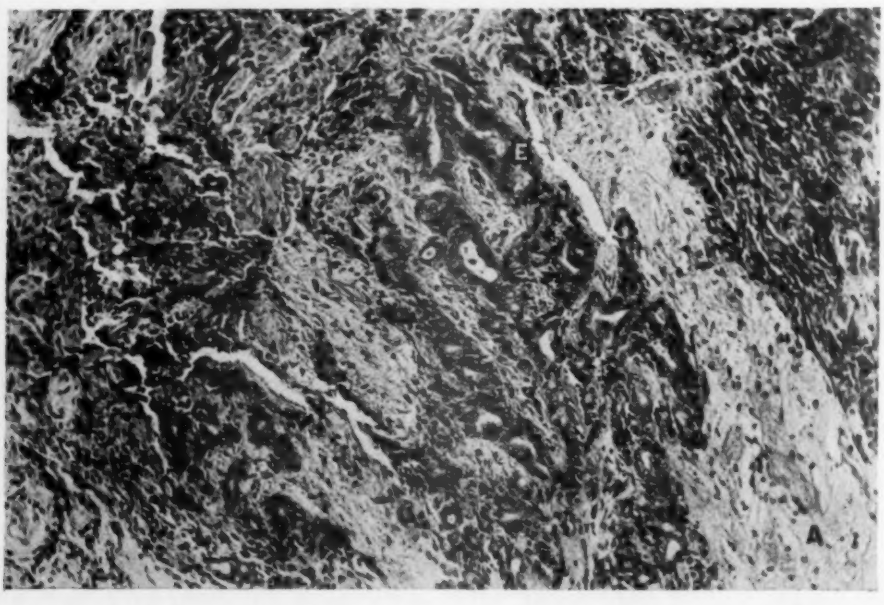

Fig. 44.—Case 14. Photomicrograph of mixed tumor showing glandular arrangement of the epithelial cells (*E*) and a myxomatous character of the stroma (*A*). (Neg. 77762, ×100.)

from enclaved cell rests consisting of two types of germ tissue, epithelium and mesenchyma. The second view is that the tumors are adenomas of the salivary glands rather than mixed tumors. It is suggested that the cartilage found in these tumors is not a true type but pseudocartilage and that the tumor epithelial cells produce mucin thus forming the ground substance of the mucinous connective tissue.

Case 14

Mixed Tumor

(Acc. 95547.) A Negro male, aged 26 years, asymptomatic, revealed radiographically on a routine examination a cystic area the size of a dime in the periapical region of the maxillary first molar (Fig. 42). No bulging of the palate was observed. A flap operation was performed and the lesion was removed. It consisted of a firm, lobulated, encapsulated structure measuring 3 by 2.4 by 2.5 cm. On cross section a thin capsule was seen from which extended interlacing fibrous septa, which enclosed nodules of gray soft tissue. Some areas presented small foci of hemorrhage.

The microscopic examination shows a well-encapsulated tumor divided into small lobes by septa. The capsule and septa consist of loose bundles of connective tissue (Fig. 43). Epithelial cells are arranged in narrow strands which seem to run in crossing palisades. In many areas, the epithelial cells arrange themselves as acini. The cells vary slightly in size, shape, and staining reaction (Fig. 44). There is a great deal of mucoid connective tissue stroma present, and also some areas of hyalinization. No cartilage or bone can be found.

Comment.—This case is an example of a mixed tumor found in the jaw as a central lesion. It appeared as a cystic lesion and only by means of a microscopic examination could it be diagnosed.

CARCINOMAS

While oral carcinomas are readily accessible, they are very malignant. Even with the most modern means of therapy, the mortality rate is extremely high. The best chance for cure lies in the early recognition of the lesion. Therefore, it is essential that one should have a thorough knowledge of the gross features and clinical course of these tumors in order to deal with them successfully. They are difficult to diagnose in their incipiency for they tend to resemble some of the inflammatory lesions of the mouth. Later they may appear either as papillary, wartlike, or ulcerating lesions. They may occur anywhere in the oral cavity; each position tends to vary the course and clinical appearance. Carcinomas which lie in proximity to bone may invade it and thus change the clinical course considerably. Most frequently seen in the oral cavity are the squamous-cell carcinomas; less frequently encountered are basal-cell carcinomas, adenocarcinomas, and transitional-cell carcinomas.

Squamous-cell carcinomas form most frequently on the lips, tongue, cheek, and palate. They form as ulcers, or papillomatous or fungating growths and metastasize early. They may start up in areas of leucoplakia. Ulcerations are common in carcinoma of the tongue, especially advanced cases, but sclerosing types are also seen. Gingival carcinoma may infiltrate the jaws in the advanced stage and therefore are difficult to differentiate from the central type. The ques-

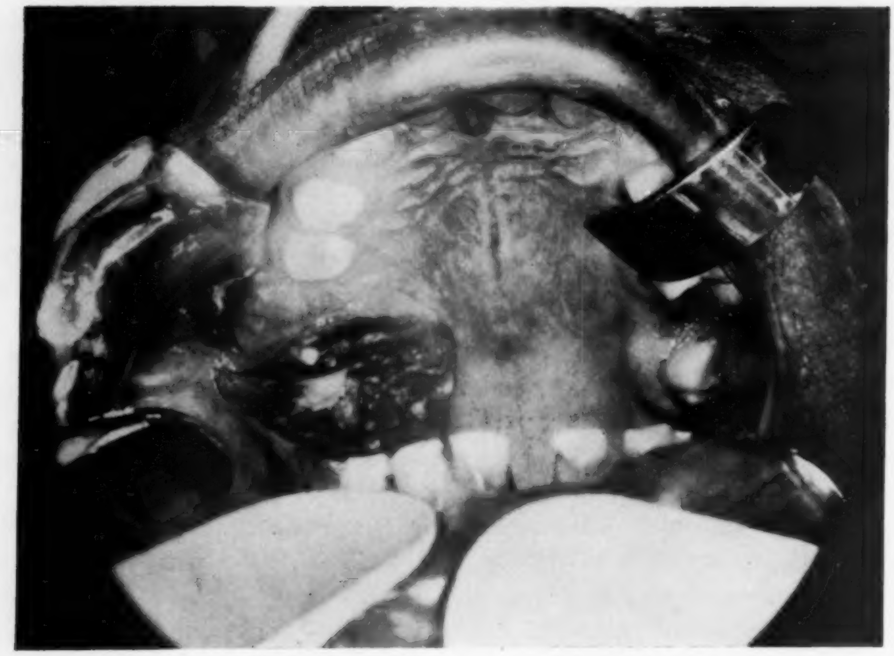

Fig. 45.—Case 15. Carcinoma, squamous cell, of palate. (Neg. 77650.)

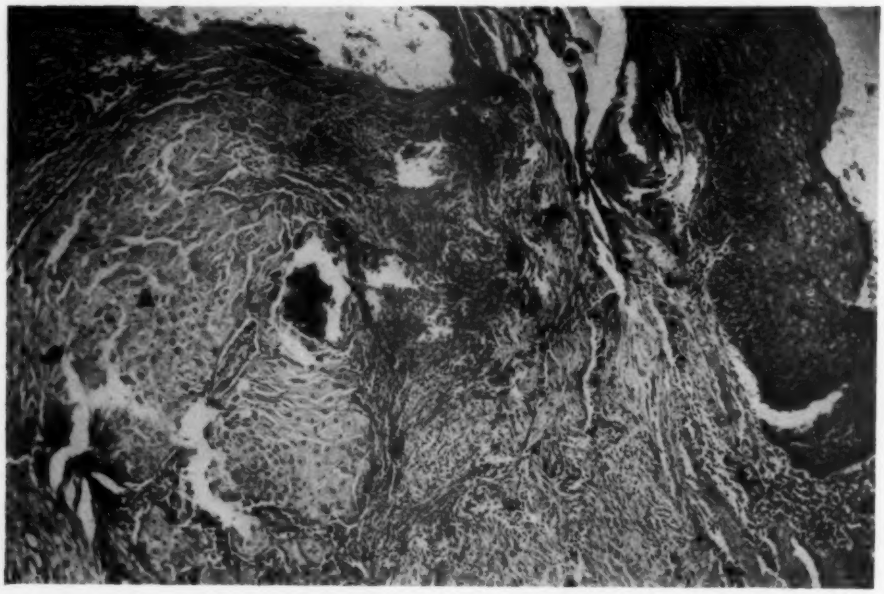

Fig. 46.—Case 15. Photomicrograph of squamous-cell carcinoma showing islands of tumor cells in the submucosa (*A*). (Neg. 77649, ×100.)

tion as to whether true central jaw carcinomas occur has been one of debate. They are said to occur from epithelial rests of tooth formation and epithelium from fissural enclavement.

Basal-cell carcinomas are far less common than the squamous type and do not metastasize, but do infiltrate. They are usually found on the upper lip and on the floor of the mouth.

The adenocarcinoma is a tumor of gland cells resembling an adenoma, but it does not retain the normal features of gland structure. The cells lose their polarity, and atypical cells are seen as well as mitoses. It usually occurs on the hard palate more than on any other part of the mouth, probably arising from the abundant glandular tissue found there. It is a slow-growing tumor and is very invasive. It usually causes a bulging of the palate which may later ulcerate. Often these tumors occur in the antrum and break through onto the palate, appearing as a painless, nontender mass.

The transitional-cell carcinoma occurs most frequently in the nasopharynx, but may involve the palate. It consists of loose hyperchromatic cells of an indistinct epithelial type that resemble the cells of reticulocytoma. The cells may lose most or even all their epithelial characteristics. They grow rapidly and are very radiosensitive; however, they recur quickly.

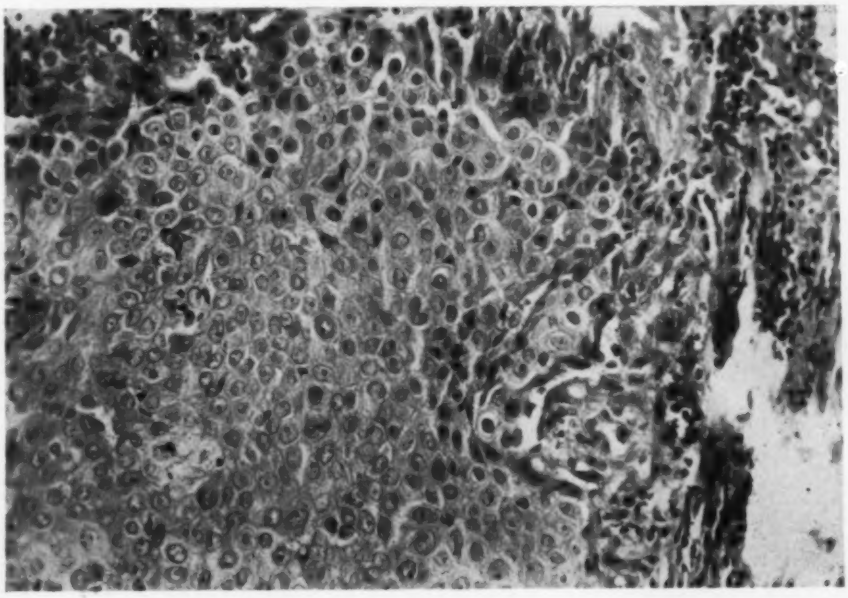

Fig. 47.—Case 15. Photomicrograph of squamous cell carcinoma, showing the character of the tumor cells. This is a high-power view of the section seen in the previous illustration at *A*. (Neg. 77139, ×350.)

Case 15

Squamous-Cell Carcinoma of the Maxilla

(Acc. 91984.) A Negro male, aged 55 years, had two loose teeth extracted two months previously and noticed that the wound did not heal. Oral examination showed a large fulminating mass in the maxillary-molar region where the teeth had been extracted (Fig. 45). The central portion showed necrosis. A biopsy was performed to determine the nature of the lesion.

The gross specimen for microscopic examination measured 1.2 by 1 by 0.4 cm. The microscopic examination reveals tissue lined by a stratified squamous epithelium. The submucosa consists of a fairly dense connective tissue in which nests of squamous cells infiltrate throughout the stroma (Fig. 46). Many of the cells were well differentiated, others are irregular in size and shape. Many mitotic figures are seen. Fig. 47 shows a high-power photomicrograph of the epithelial cells.

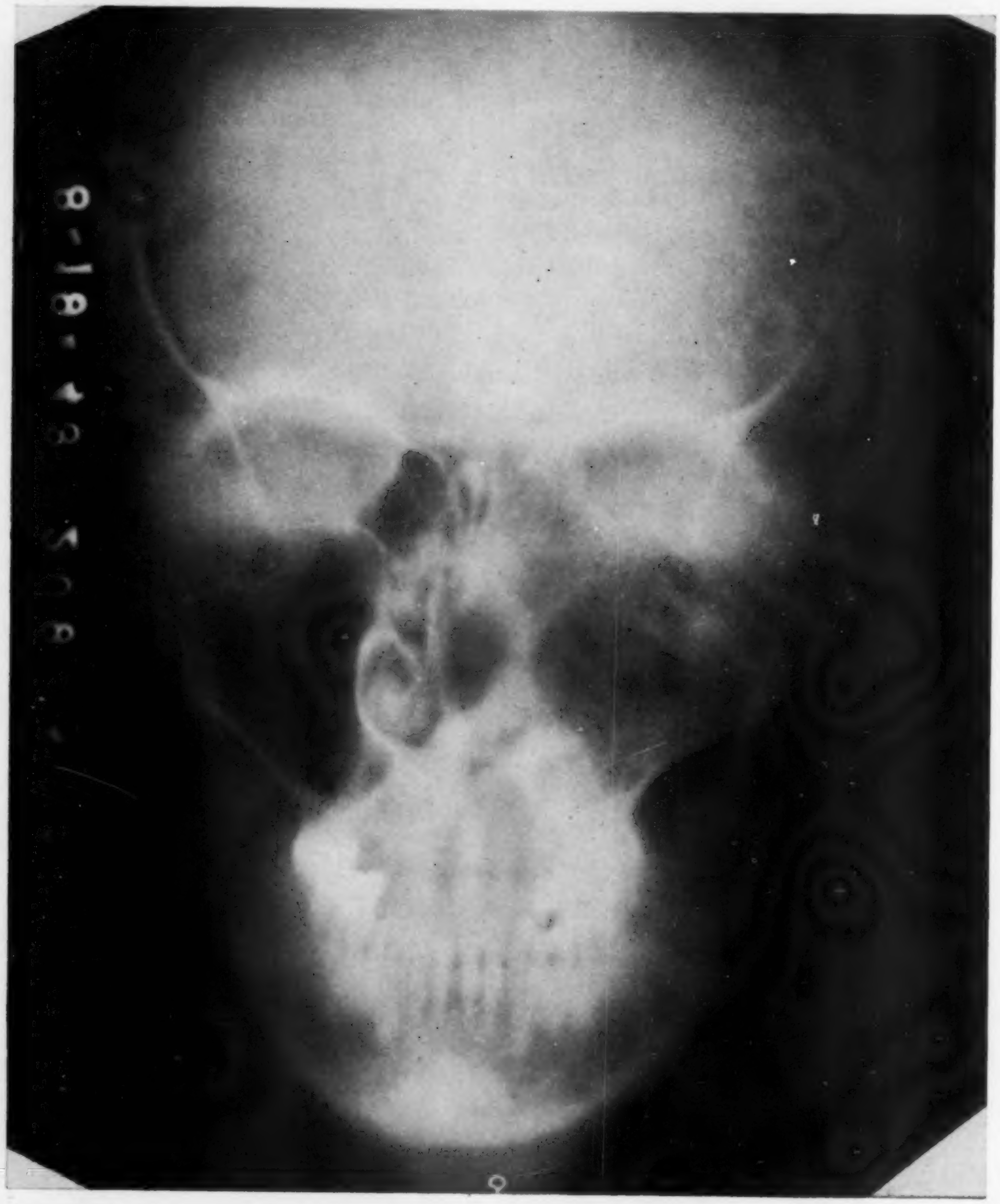

Fig. 48.—Case 16. Radiograph of adenocarcinoma of antrum showing a cloudy appearance and destruction of the walls. (Neg. 76705.)

Comment.—This case illustrates the danger of removing loose teeth without having first made a radiographic examination. Had one been done, treatment could have been instituted immediately with perhaps a little better prog-

nosis, although the prognosis was bad even at the time of the extraction of the teeth.

Case 16

Adenocarcinoma of Antrum

(Acc. 97238.) A white male, aged 40 years, complained of repeated attacks of suppurative sinusitis. The past history otherwise was not pertinent. A radiographic examination showed a marked cloudy appearance of the left sinus (Fig. 48). The turbinates on that side appeared to be involved also. A

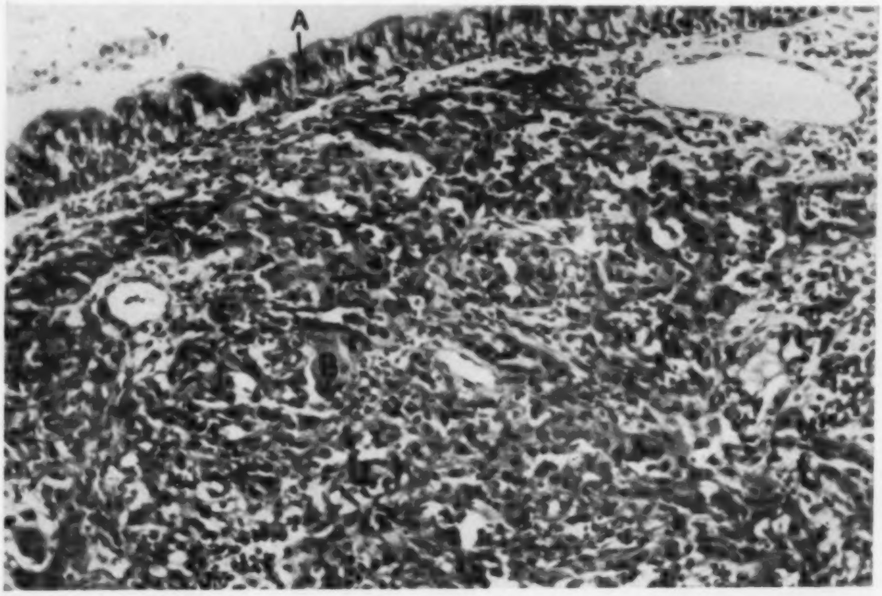

Fig. 49.—Case 16. Photomicrograph of adenocarcinoma showing the tumor lying in the submucosa (*B*). The normal respiratory epithelial lining is seen at (*A*). (Neg. 7760, ×240.)

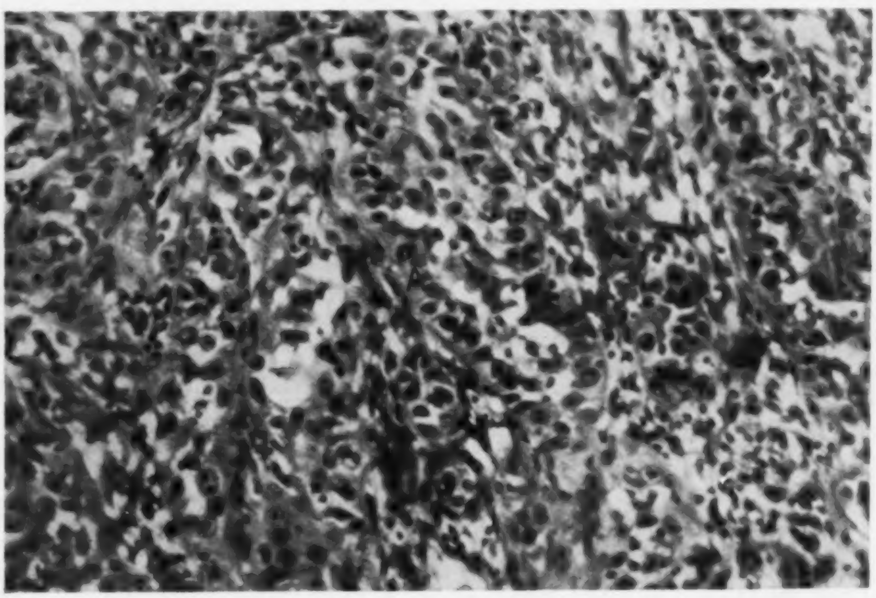

Fig. 50.—Case 16. Photomicrograph of adenocarcinoma showing the glandular arrangement of the tumor cells (*A*). (Neg. 77761, ×350.)

Caldwell-Luc operation was performed and the mucous membrane and polyps, consisting of irregular, firm, tough, yellowish-white tissue, were removed.

The microscopic examination reveals a tissue packed with tumor cells, generally in form of small acini, but sometimes growing in irregular fashion in short strands and cords, permeating the connective tissue stroma (*B*, Fig. 49). The tumor cells show moderate variation in size and depth of staining of the nuclei and many bizarre and giant nuclei with mitoses. The cytoplasm is palely basophilic, generally columnar, with many fine vacuoles, suggesting active secretion. The epithelium lining the mucosa of the antrum can be seen at *A* of Fig. 49. The acinic structure of the tumor can be seen at *A* of Fig. 50.

Fig. 51.—Case 17. Radiograph of transitional-cell carcinoma. A marked osteolytic area is seen in which a supernumerary tooth is present, giving the lesion the appearance of a dentigerous cyst. (Neg. 77652.)

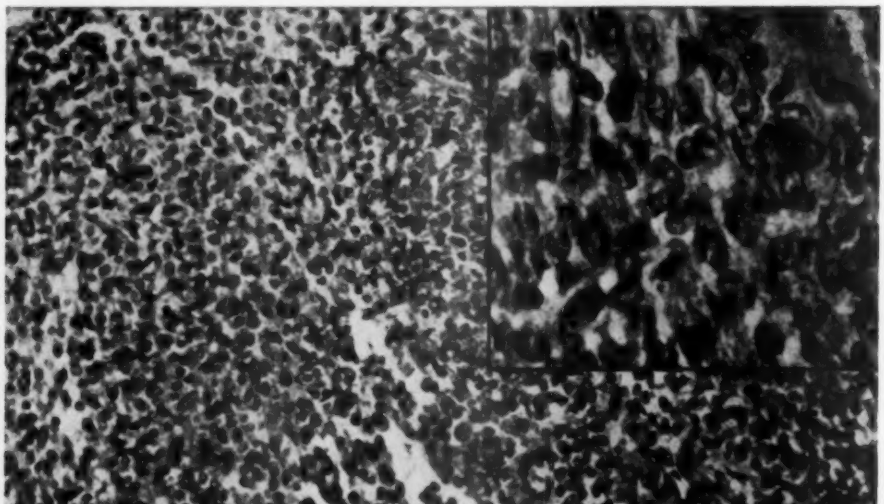

Fig. 52.—Case 17. Photomicrograph of transitional-cell carcinoma showing the cellular structure of the neoplasm. (Neg. 77654, ×280.) (Insert, Neg. 77375, ×765.)

Case 17

Transitional Cell Carcinoma of Maxilla

(Acc. 86153.) A white male, aged 26 years, had had a maxillary canine extracted in 1936, and had been troubled with the area since. In January,

1942, a moderate swelling began in the left cheek and persisted for two months, at which time it was incised and drained. Radiographs taken in June, 1942, showed a marked rarefaction of the entire maxilla from the lateral incisor area to the first molar. A supernumerary tooth in a large cyst having the appearance of a dentigerous cyst was evident. Fig. 51 illustrates this lesion in the dental films.

The microscopic examination of the biopsied specimen reveals sections composed of large amounts of necrotic hemorrhagic material in which there are masses of embryonic epithelial cells containing large vesicular nuclei with conspicuous nucleoli (Fig. 52). Many of these cells show mitoses. The cytoplasm is pale and the cell outline not distinct. In some areas there is a slight palisading around vascular channels, while in other areas the cells are arranged in cords and suggest glandular formation. In these latter areas the cells approach the low columnar type. One section shows dense fibrous tissue covered with stratified squamous epithelium. In this fibrous stroma there are a few masses of neoplastic cells which centrally show considerable keratinization.

Comment.—This tumor is found in what seemingly, from x-ray examination, was a dentigerous cyst. It is possible that the tumor arose from the epithelium in the cyst. Again, the importance of microscopic examination of tissue in such lesions is stressed.

Case 18

Transitional-Cell Carcinoma

(Acc. 70922.) A white male, aged 20 years, complained of an ulcer on the palate and general malaise. The past history revealed that, about a month previously, the patient had had a slight swelling and pain on the left side of his face in the region of the maxillary third molar. A tooth was extracted, no anesthetic being used; the patient experienced practically no pain. The socket was cleansed daily with an antiseptic. A few days later, an ulcer developed in the palate, and the lymph nodes at the angle of the left jaw became swollen, but not painful or tender. They increased in size and a large node developed below the left ear. The ulcer of the palate grew larger and deeper and extended onto the lateral side of the maxilla over the molar teeth (Fig. 53).

On physical examination, the patient appeared undernourished and anemic. He weighed 116 pounds, his normal weight being 135 pounds. The anterior and posterior cervical lymph nodes on the left side, the anterior cervical nodes on the right side, and the left axillary nodes were all enlarged. The latter could easily be seen when the patient raised his arm. The nose appeared patent and was lined with a normal mucous membrane. The tonsils and larynx appeared normal, and no nasopharyngeal growth could be detected. The mouth showed a large, deep, granular ulceration of the soft and hard palate extending medially from the last molar. A hard tumorous growth in the maxillary mucobuccal fold of the left side in the region of the molar teeth was evident. Transillumination showed the frontal sinuses to be clear, but a slight hazy appearance of the right antrum was noted.

A biopsy was taken and a diagnosis of transitional-cell carcinoma was made. The patient was given x-ray treatment. He ran a low-grade fever and became more emaciated and cachectic. One morning, he developed suddenly a

massive hemorrhage from the ulceration on the palate. This was stopped, but later that day he developed another one and died.

Autopsy examination was negative except for the palate, lymph nodes, and a tumor mass in the left supraclavicular region. The latter was comprised of moderately firm, slightly necrotic, somewhat hyaline homogeneous tissue. A tumor mass was also found in the anterior wall of the stomach, and three small nodes in the small bowel.

Fig. 53.—Case 18. Ulcerative lesion of the palate which upon microscopic examination proved to be a transitional-cell carcinoma. Notice the submandibular swelling; this was due to metastases to the lymph nodes. (Neg. 77756.)

Microscopic examination of the tissue of the palate revealed a section lined on one surface by a stratified squamous epithelium (*B*, Fig. 54) showing a marked acanthosis and some parakeratosis. One end was ulcerated, the base consisting of a dense inflammatory infiltrate (*A*, Fig. 54). The submucosa consisted of a loose to moderately dense connective tissue in which there was a dense accumulation of inflammatory cells composed chiefly of lymphocytes, plasma cells, and polymorphonuclear leucocytes. Deep in the submucosa and nearer the surface at the area of the ulcer, a tumor mass was present consisting of cells, epithelial or epithelioid in character with large clear cytoplasm and having the cohesiveness of epithelium. The nuclei varied in size and staining reaction (Fig. 55). Infiltrating through the tumor mass were numerous inflammatory cells, and bordering the former tissue numerous multinucleated giant cells were present.

Fig. 54.—Case 18. Photomicrograph of transitional-cell carcinoma. The normal epithelial covering can be seen at *B*, while at *A*, ulceration of the surface is evident (Neg. 77562, ×10.)

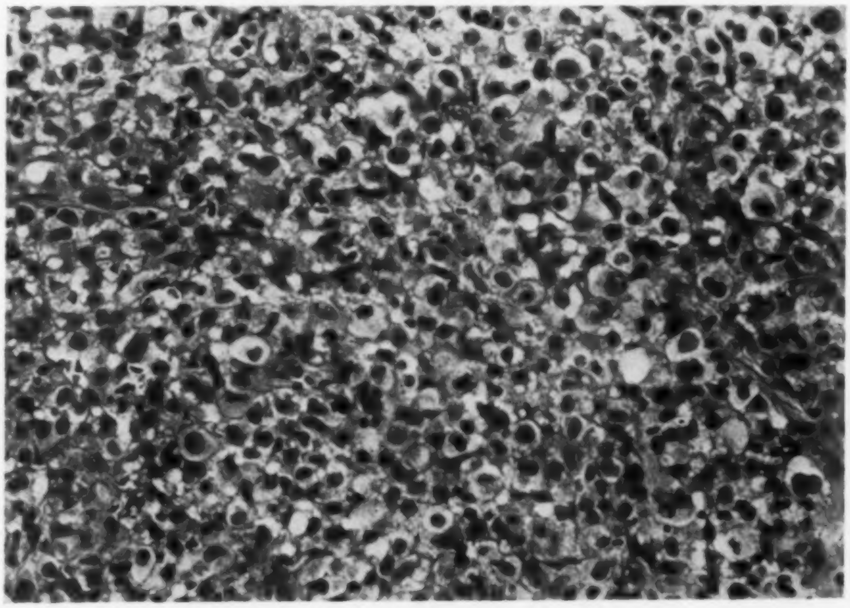

Fig. 55.—Case 18. Photomicrograph of transitional-cell carcinoma showing the tumor cells. (Neg. 77141, ×435.)

The diagnosis of transitional-cell carcinoma was confirmed.

Comment.—This case is a typical example of a group of tumors seen in the palate and nasopharynx which metastasize very early to the lymph nodes. This type of tumor is at times difficult to differentiate from an anaplastic squamous-cell carcinoma and from reticulocytoma, but its clinical course is characteristic and it is quite radiosensitive. It is also called "lymphoepithelioma" because of the usual association of lymphatic tissue and epithelium.

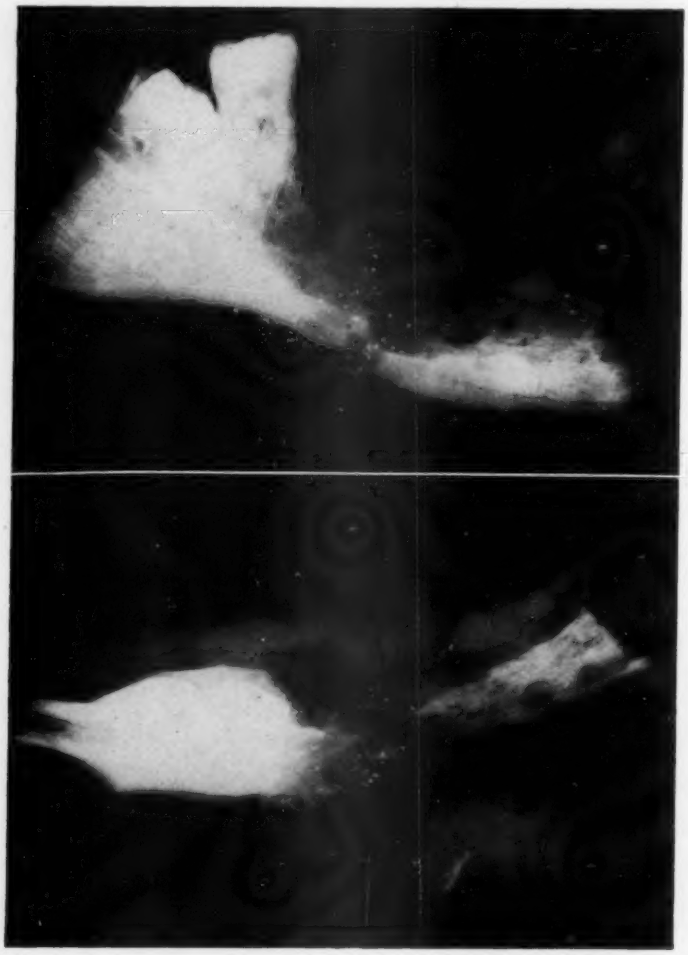

Fig. 56.—Case 19. Radiographs of the resected portion of the mandible, the site of an undifferentiated carcinoma. (Neg. 77757.)

Case 19

Undifferentiated Carcinoma

(Acc. 81625.) A 21-year-old male was admitted to the hospital with a past history of extraction of two loose teeth and subsequent pain and swelling in the mandible. The area did not heal and a biopsy was taken revealing an anaplastic squamous-cell carcinoma. He was given x-ray therapy over a period of ten days. Upon physical examination, he was found to have a large fungating, ulcerated neoplastic mass involving the right alveolar region and floor of the mouth.

After removal of as much of the necrotic tissue as possible, examination showed a large ulcer, with a floor of greenish-gray tissue, extending from the right lateral cheek at the level of the usual occlusion line, downward across the floor of the mouth, to the inner surface of the left side of the mandible. The mucosa over the left part of the mandible remained intact. The ulcer extended

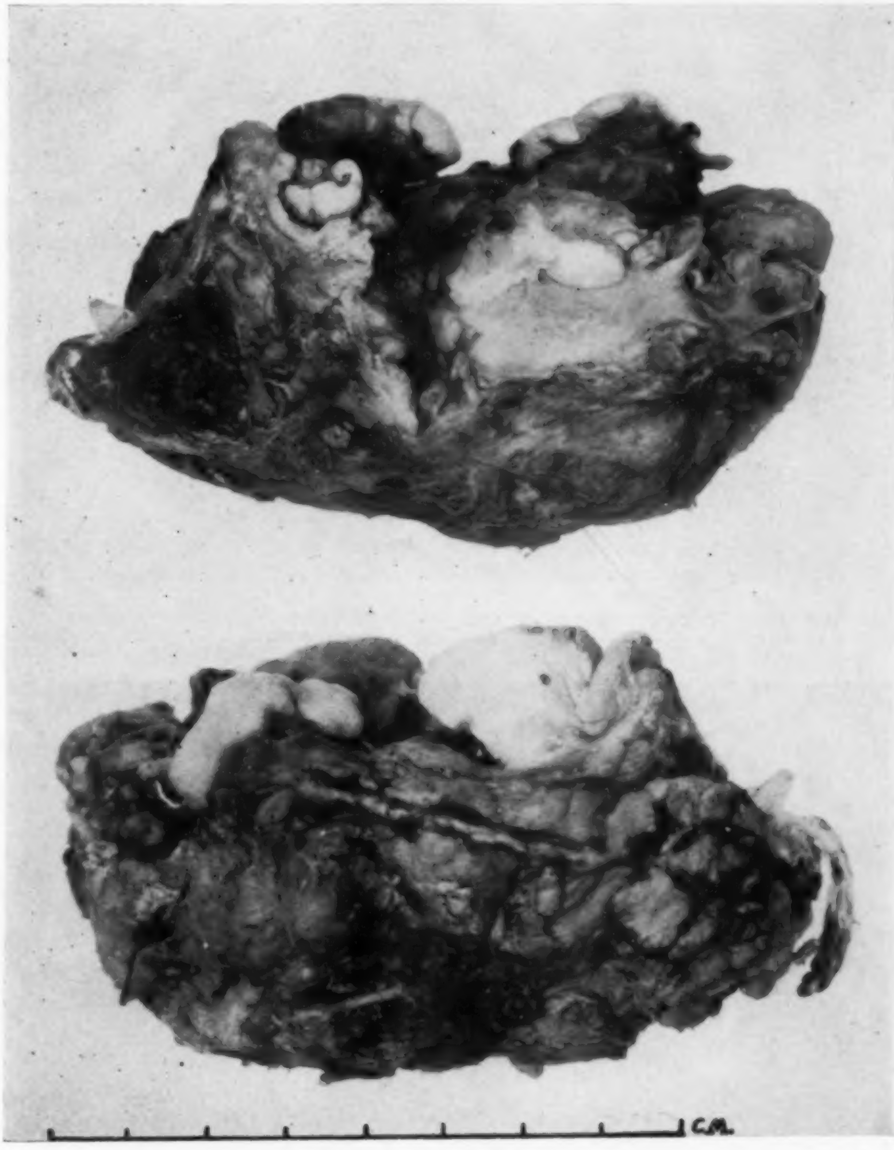

Fig. 57.—Case 19. Photographs of the tumor mass around the mandible. (Neg. 77757.)

backward from the posterior surface of the mandible in the midline to the base of the tongue. The tongue was pushed upward slightly. The ulcer also extended on the right side almost to the mucocutaneous margin of the lower lip. The inferior portion of the tongue was indurated and slightly nodular. The lower lip in the midline was firm and slightly nodular.

A combination of surgery and x-ray therapy was thought to be the best means of treatment, and prior to operation, the patient was given extensive x-ray therapy for twenty days. The tumor of the jaw regressed considerably after this treatment and a month later a resection was performed of the right mandible with excision of the tumor extensions into the mouth. Radiographs

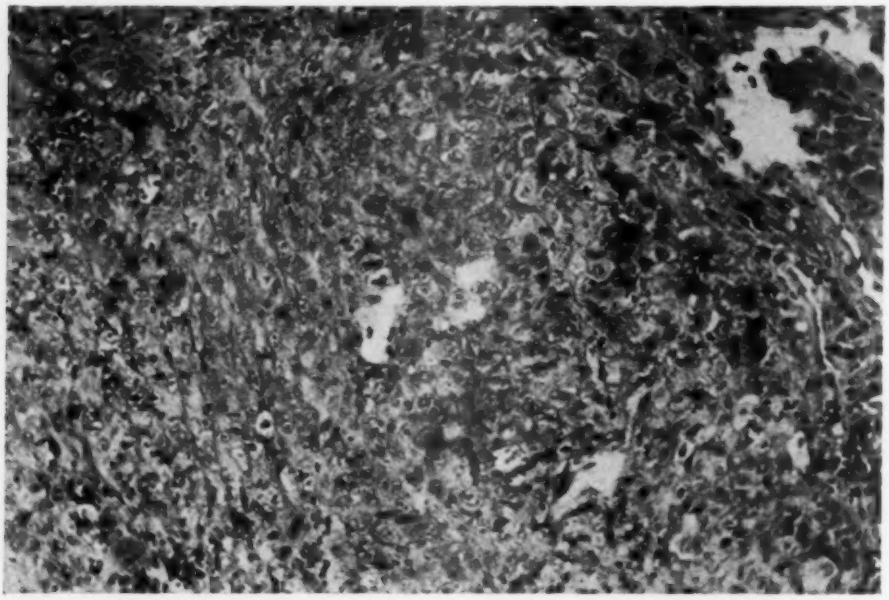

Fig. 58.—Case 19. Photomicrograph of the undifferentiated carcinoma seen in the two previous illustrations. The pleomorphism of the cells is evident. (Neg. 77759, ×350.)

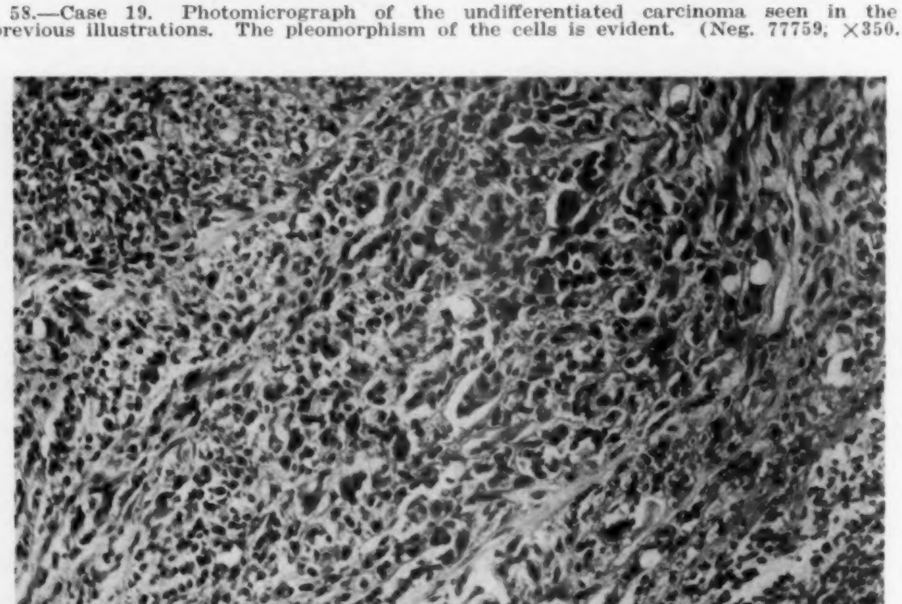

Fig. 59.—Case 19. Photomicrograph of another section of the undifferentiated carcinoma showing a different character of the cells. (Neg. 77758, ×165.)

of the resected portion can be seen in Fig. 56; the resected specimen can be seen in Fig. 57. His immediate postoperative course was excellent and his recovery was uneventful.

However, about three weeks after operation, the tumor recurred and proliferated very rapidly into the adjacent soft tissues of the mandible. The patient began to go progressively downhill. He received more x-ray therapy without avail, and his clinical course was manifested by irregular periods of fever and marked emaciation resulting chiefly from his inability and lack of desire to eat. The patient died in two months.

Microscopic examination of sections taken from several areas in the resected part of the mandible reveal the tissue to be composed of irregular sheets of neoplastic epithelial cells separated by a variable amount of fibrous stroma. The cells are poorly defined, generally polygonal, and have large vesicular nuclei with distinct nucleoli (Fig. 58). Some fine strands of fibrous tissue separate these cells, but the demarcation is poor. Mitotic figures are quite numerous and atypical. In a few regions the cells are spindle-shaped and arranged in solid bundles. The surfaces are ulcerated and necrotic. Along one edge there is some dense fibrous tissue showing some invasion by the neoplastic cells. Along this invaded zone, foreign body giant cells are present. In many areas the sections resemble an osteogenic sarcoma (Fig. 59).

SARCOMA

A sarcoma is a tumor of connective tissue origin, retaining more or less of the characteristics of the specific type, but endowed with a power of invasion and metastasis. It is apt to occur at any age, but it has a higher frequency during the first few decades of life. It commonly metastasizes by way of the blood stream, the lung being the most frequent location for secondary tumors. It may be differentiated so that its tissue of origin is recognizable, for example, fibrosarcoma, liposarcoma.

Case 20

Reticulum-Cell Sarcoma

(Acc. 96206.) A white male, aged 24 years, noticed some nasal congestion about two months before hospitalization. This lasted about a month when he began to note an unusual soreness and swelling on the left side of his palate. At the time of admission, a large necrotic ulceration was noted on the left half of the soft palate, measuring 2.5 by 3.5 cm. in diameter and extending onto the left hard palate. There was some bleeding and some sponginess of the gingiva of the left maxilla. A diagnosis of Vincent's stomatitis and gingivitis was made. He was treated locally with mapharsen and hydrogen peroxide, but the condition became worse.

The soft palate became edematous, and the right side became involved. In a few days, the ulceration spread down into the pharynx and started to involve the left faucial tonsil. It also spread up into the nasopharynx behind the palate. At this time, he was seen by several consultants, and it was their unanimous impression that this was probably a lympho-epithelioma (transitional-cell carcinoma). The condition spread further rapidly and it was noted that his anterior nasal septum was becoming thicker, although no ulcerations were seen in either nares. The nasal passageways were mere vertical slits and the septal walls felt spongy or rubberlike in consistency. No nasal ulcerations

Fig. 60.—Case 20. Photograph of reticulum-cell sarcoma of palate. A diffuse fungating lesion is evident. (Neg. 77754.)

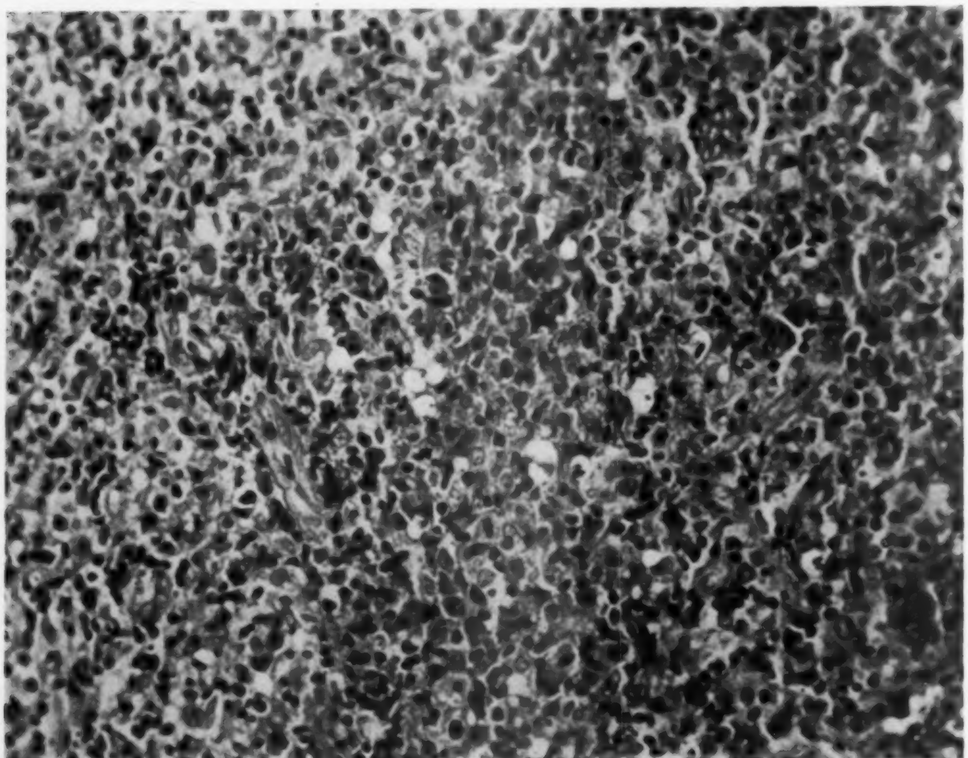

Fig. 61.—Case 20. Photomicrograph of reticulum-cell sarcoma showing the character of the tumor cells. (Neg. 77755, ×165.)

could be seen. The paranasal sinuses appeared clear on transillumination. The entire hard and soft palate, on both sides, both tonsils, and the left pharyngeal and hypopharyngeal wall were involved in a rapidly growing ulcerative and tumor process (see Fig. 60). In some areas, the tissue appeared ulcerated, in other regions, the tissue appeared granulomatous, and in still others, it had the appearance of a typical fungating or diffuse papillomatous lesion. The uvula was also ulcerated and plastered down against the right palatal margin. The right hypopharynx was moderately involved, but not as severe as the left side. The epiglottis appeared involved only at the insertion. The larynx and pyriform sinuses were not involved. There was some dysphagia, but no dyspnea. Several small soft nodes could be palpated in each anterior cervical chain but there was no general lymphadenopathy and no evidence of any distinct metastases. The remainder of the physical examination was essentially negative.

Microscopic examination of a biopsy specimen reveals a stratified squamous epithelium with marked pseudo-epitheliomatous hyperplasia covering the surface. The submucosa is densely infiltrated by neoplastic cells which appear lymphoid in character and vary in size. They are generally round to polygonal in shape, with a pale cytoplasm and indistinct outline. Mitoses are very frequent. The nuclei are basophilic; the nucleoli are small but prominent (Fig. 61). There is a marked inflammatory infiltrate in the tissue, with polymorphonuclear leucocytes and lymphocytes predominating.

The tumor is a reticulum-cell sarcoma.

The patient received eighteen x-ray treatments, 2,400 r. to portal right neck, the same dosage to the portal left neck, and a similar dose to the portal anterior face, making a total dosage of 7,200 r. The tumor melted away rapidly, and to date has shown no recurrence, despite the fact that slight ulceration on the palate remains. This has been attributed to radiation necrosis.

Comment.—This case illustrates the danger of a misleading diagnosis of Vincent's infection. The latter should not be accepted until the possibilities of an underlying, more serious condition have been eliminated.

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Editorial

The Army Medical Museum Number

The Army Medical Museum was established in 1862 by order of Surg. Gen. William A. Hammond. In his book, *Victories of Army Medicine*, Brigadier General Hume* says that Hammond had in mind the study of specimens collected in order to "lead to reduction in mortality." Seven months after opening the Museum, 1,349 specimens had been collected. According to a recent article by Howard T. Karsner, M.D.,† published in the *Journal of the American Medical Association*, visitors from Europe have repeatedly praised the Museum. Berenger Ferand, in 1870, said that the United States had done as much in five years as had all Europe in a century, and that our Museum contained more specimens than all the pathologic anatomic museums of Europe combined.

Today, there are on file large collections, even of rare diseases, such as 2,000 cases of melanoma of the eye, 41,000 tumors of the bladder, and 70 adamantinomas of the jaws. These are available for reference and study by properly qualified investigators.

Under the authority of the Surgeon General, the Museum has instituted a system of resident consultants who take up residence in Washington from two to three weeks, during which time they are in daily attendance. This gives them an opportunity to see pathologic material in volume and variety, such as cannot be equaled in any other pathological laboratory in the world.

The institution is closely associated with two other great institutions, the Army Medical Library and the Army Medical School, and the Museum staff has been responsible for teaching pathology in the school. Under the supervision of Colonel James E. Ash, Curator, the Army Medical Museum sponsors twelve registries. Certain officers have been assigned to develop various special fields, such as neuropathology, dermal pathology, and oral pathology. The Registry of Dental and Oral Pathology is sponsored by the American Dental Association, with Dr. Henry Swanson of Washington, D. C., as chairman of the National Museum Committee. The Registry offers a number of valuable services to the profession.

Anyone may send cases to the Registry. However, the latter does not act as a diagnostic laboratory in competition with civilian pathologists, but an opinion will be rendered. A group of consultants has been appointed, to which are submitted unusual and bizarre cases, or those in which a controversy exists. The board of consulting pathologists of the Registry of Dental and Oral Pathology is as follows: Drs. Kurt H. Thoma, Paul E. Boyle, Lester Cahn, Hamilton B. G. Robinson, Donald Kerr, and Balint Orban. In 1943 there was a substantial increase in the number of cases diagnosed, because of the great

*J. B. Lippincott Co., 1943.
†J. A. M. A. 124: 710, 1944.

amount of material sent in by members of the Dental Corps of the Army. Approximately 3,500 cases have been registered.

Study or loan sets are sent out to individuals or schools throughout the country. These sets contain about 100 slides of the various phases of oral pathology, each slide being described in a syllabus.

An Atlas of Dental and Oral Pathology is published to furnish a fundamental teaching and reference guide for the student and practitioner.

State boards are furnished, upon request, with pathologic material for examinations in oral pathology, which should stimulate the teaching of dental and oral pathology in all dental schools.

Kodachrome slide sets of local and generalized processes involving the mucous membrane and tongue, and other dental and oral lesions, are available for study. Sets of thirty slides on such subjects as caries, periodontal disease, stomatitis, tumors, and jaw lesions are available.

The Army Medical Museum Number is a new venture, and we are indeed fortunate to present it to the readers of the JOURNAL. A great deal of credit is due Major Joseph L. Bernier, D.C., U.S.A., who started these valuable contributions, and to Lt. Henry M. Goldman, D.C., A.U.S., who succeeded him as Secretary and is now Pathologist to the Registry of Dental and Oral Pathology at the Army Medical Museum. Lieutenant Goldman is certainly to be congratulated for continuing this very excellent project with untiring energy and a fine scientific spirit that promises much for the future.

K. H. T.

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Original Articles

GIANT CELL TUMORS OF THE JAWS

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GIANT cell tumors of the jaw are connective tissue tumors arising within the bones or from the periosteum.

Two types are recognized; the central benign giant cell tumor having its origin within the bone and expanding in all directions, eventually breaking through the cortex but generally leaving the periosteum intact; and the peripheral giant cell tumor arising from the periosteum, expanding superficially into the bone and peripherally into the mucous membrane.

Whether these tumors are neoplasms or inflammatory processes has not been fully determined. The tendency to recur following incomplete eradication, the absence of encapsulation, and the destruction and resorption of tissues with which they come into contact during their expansile growth are neoplastic evidences.

Microscopically, the tumor tissue is composed of elements found in any inflammatory processes; small round cells and spindle-shaped cells. However, the large number of multinucleated giant cells found in the tumor tissue gives rise to the question of their significance.

One form of giant cell is the biologic reaction to the presence of foreign bodies in the tissues. Classic examples are the tissue reactions around absorbable suture material, such as catgut. Microphages fuse to form large protoplasmic masses in which the cell boundaries disappear and the nuclei continue to divide. The action of these cells on the catgut results in its absorption.

Is the initial process in the formation of the giant cell tumor that of a foreign body reaction? If so, why doesn't it arise at the site of bits of amalgam often accidentally left in sockets following extraction?

Are these giant cells, osteoclasts resorbing the bone with replacement by small round and spindle-shaped cells? If they are, is there a relationship between the solitary giant cell tumors of the jaws and hyperparathyroidism?

There are numerous reports of *multiple* giant cell tumors involving one or more bones which are definitely part of the picture of osteitis fibrosa cystica, in which removal of a parathyroid adenoma is followed by spontaneous cure.

The *Archives of Clinical and Oral Pathology,* June, 1937, cites a case in which excision of a giant cell tumor of the jaw was followed by recurrence in a different location. Subsequent examination disclosed a parathyroid adenoma and multiple cysts in other bones. Excision of the adenoma was followed by spontaneous healing.

In these cases there is increased calcium and decreased phosphorous in the blood serum. The phosphatase is increased. Osteoporosis takes place, with subsequent cyst formation. Occasionally, one or more osteoclastomas

form, and it is these tumors which microscopically resemble the solitary giant cell tumors of the jaws.

That the giant cells of the solitary tumor exhibit a foreign body reaction is evidenced by the clear-cut destruction of apices and even lateral surfaces of tooth roots with which they come into contact.

A consideration of giant cell tumors arising within the bone necessitates study to determine whether a tumor is part of a general disturbance or exists alone. Blood calcium and phosphorous determination, and x-rays of both long and flat bones must be studied.

The *etiology* of the central benign giant cell tumor is obscure. The cause most frequently advanced is trauma. Experimentally, however, trauma has not been followed by tumor formation in man.

Hemorrhage within the bone has been considered, the tumor resulting as a process of repair.

Localized areas of calcium resorption with endothelial proliferation has been said to be a cause.

X-ray diagnosis will depend on proper interpretation. The involved areas are radiolucent and cystlike in character, but no lamina dura is present. At times, fine bony septi may be seen traversing through the radiolucent mass.

The writer has found one constant sign occurring in almost all of his cases. This sign is a varying gradation in the density of the mass. From the most radiolucent area in the center, the density increases until it becomes radiopaque and merges with the surrounding normal bone. This seems to correspond to the amount of bone destruction, the greater radiolucency being at the point of the original focus.

The treatment is complete eradication by excision and curettage, followed by cauterization. Incomplete eradication results in recurrence. Metastasis to the lungs has been reported following recurrence. X-ray therapy has been found favorable by some.

The *peripheral* giant cell tumor is one of the epuli. The term epulis has the general meaning of a tumor of the gums. The three common types recognized are angioma, fibroangioma and benign giant cell tumor. A fourth type may be added to the classification of epulides in the recognition of pregnancy tumors, some of which disappear following term, and others which must be removed surgically.

The peripheral giant cell tumor arises from the periosteum, penetrates the mucosa to appear in the mouth in sizes ranging from a pea to an orange. During its growth, the bone on which it lies becomes involved, first superficially and later much more deeply.

When it occurs between teeth, it causes the latter to become markedly separated. If the tooth roots are within the mass, well-defined punched out areas through the cementum and dentine may occur.

The peripheral giant cell tumor may be differentiated clinically at times by its color and compressibility. It has a bluish hue and is nodular in texture. It may or may not turn whitish on pressure, depending on its vascularity.

The angioma has a reddish color, and when compressed becomes whitish. When the pressure is released, it immediately becomes red.

The fibroangioma is more firm and pinkish in color.

CASE REPORTS

CASE 1.—A. B., male, aged 26 years, developed a swelling in the lower anterior jaw, for which his dentist extracted a lower left central incisor. This was followed by a discharge of pus from the socket. The socket failed to heal and he was referred for consultation.

Examination.—An open lower left central socket was disclosed through which there protruded a mass of granulation tissue. No pus could be expressed.

X-Ray Examination.—There was revealed what appeared to be a circumscribed radiolucent mass at the apex of the socket, consistent with a diagnosis of cystic granuloma. Below the radiolucency, a circumscribed, more radiopaque area could be seen. This area was homogeneous and bone trabeculae were present at the periphery. There was a variation in the density of the involved area (Fig. 1).

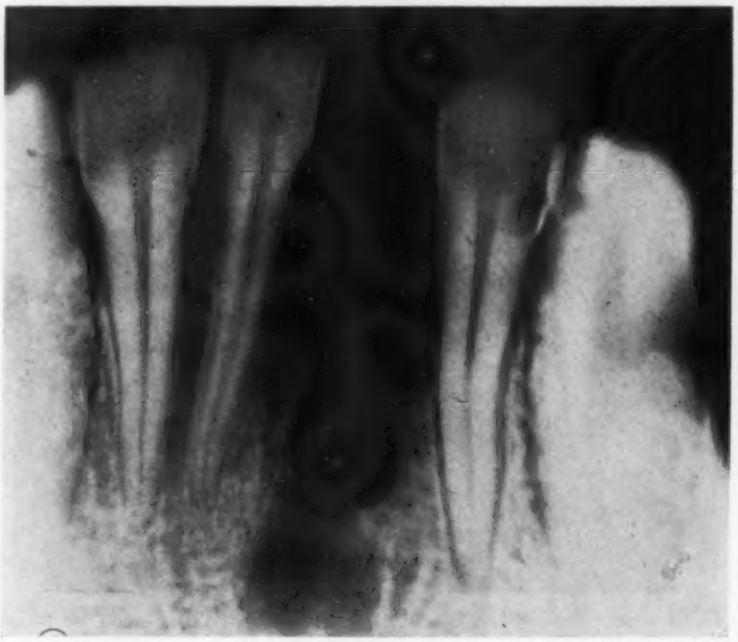

Fig. 1.—Beginning giant cell tumor below cystic granuloma. Note variation in density in the tumor mass.

Operation.—An open operation was performed. The cystic granuloma was enucleated, below which there appeared a more or less solid mass, dark reddish-brown in color. The consistency was that of congealed jelly. This was removed and the bony cavity cauterized. The tumor measured 0.5 cm. in all diameters.

Microscopic Diagnosis.—Central benign giant cell tumor. This was one of the smallest giant cell tumors in the writer's series.

CASE 2.—A. W., female, aged 29 years. Two years previously she had fallen on her back and the impact had forced the lower jaw against the upper. Two months ago, she began having pain in the lower right jaw and noticed an enlargement.

Examination.—Examination disclosed a definite bulge of the buccal cortex extending from the lower right cuspid to the second premolar, and from below

the gingiva to the mucobuccal fold. Pressure on this area resulted in crepitus. The teeth were vital to electrovitality tests. Blood calcium and phosphorous were within normal limits.

X-Ray Examination.—X-rays of the long bones were negative. X-ray examination of the jaws disclosed an irregular radiolucent area extending from the lower right lateral apex to the mesial of the lower right second premolar. The area was below the apices of the teeth. There was variation in the density of the involved bone. The periphery of the area was radiopaque and merged with the normal bone markings. The greatest radiolucency was in the anterior portion. Through some parts, bone trabeculae could be made out (Fig. 2).

Fig. 2.—Giant cell tumor. Note absence of lamina dura and variation in the density of the involved bone.

Operation.—An open operation through the buccal plate was performed and a jellylike, nonencapsulated mass was removed. The bony cavity was cauterized.

Microscopic Examination.—Microscopic examination showed an outer shell of bone whose trabeculae were separated by a cellular connective tissue. Within was a cellular, very vascular, somewhat edematous tissue in which numerous multinucleated giant cells of epulis type were present. A rare mitotic figure was seen.

Diagnosis.—Central benign giant cell tumor of mandible.

Comment.—No x-rays of the teeth or jaws were available to determine whether the mandible had been involved before her accident.

Case 3.—R. A., male, aged 47 years. The patient had a lower left first premolar extracted about seven weeks previously, following which his left lower jaw swelled. His dentist made an incision through the mucous mem-

brane for drainage, but no pus was obtained. For seven weeks, he had had intermittent pain in this jaw without relief.

Examination.—There was marked enlargement of the left mandible. A left third molar and left lateral incisor were present. Between these two teeth, the jaw was edentulous. Extending from the molar to the lateral incisor was a large semisolid mass. The buccal and lingual plates could not be felt, except at the lower borders. The mucous membrane over the tumor had a bluish hue. There were no palpable submandibular glands.

Blood calcium and phosphorous were within normal limits.

Fig. 3.—Giant cell tumor. Note variation in the density of the involved bone.

X-Ray Examination.—X-rays of the long bones were negative. X-ray examination of the left mandible disclosed a large radiolucent area extending from the molar to the lateral. There was a variation in the density with peripheral radiopacity merging with normal bone. The mass appeared to be divided by a fine sweeping bony septum giving the appearance of a multilocular cyst. There was no lamina dura present around the mass (Fig. 3).

Operation.—The tumor was removed and the bony cavity cauterized.

Microscopic Diagnosis.—Central benign giant cell tumor.

CASE 4.—E. G., female, aged 35 years. The patient had been wearing an upper artificial denture for several years. About one year ago, she noted a growth in the upper left jaw, which had been progressively increasing in size. There was no pain except when wearing the denture.

Examination.—Examination disclosed a tumor mass in the left maxillary premolar area about the size of a walnut, somewhat nodular in character. The

mucous membrane over the mass was bluish in color. On its upper border was an ulcerated fissure corresponding to the flange of the denture. On palpation, there appeared to be a loss of both buccal and palatal bone. On each side of the tumor were flabby hyperplastic masses. The blood calcium and phosphorous were within normal limits.

X-Ray Examination.—X-rays revealed an irregular loss of bone in the upper left jaw. In places bone trabeculae could be seen traversing the radiolucent area (Fig. 4).

Operation.—The tumor was removed and the bony cavity cauterized.

Microscopic Diagnosis.—Peripheral benign giant cell tumor.

Comment.—This case is one in which it is difficult to determine whether the tumor originated within the bone or from the periosteum. Bone loss was much more extensive than that generally caused by the peripheral type of tumor. Nevertheless, the progressive increase in size within the mouth favors the diagnosis of peripheral benign giant cell tumor of the maxilla.

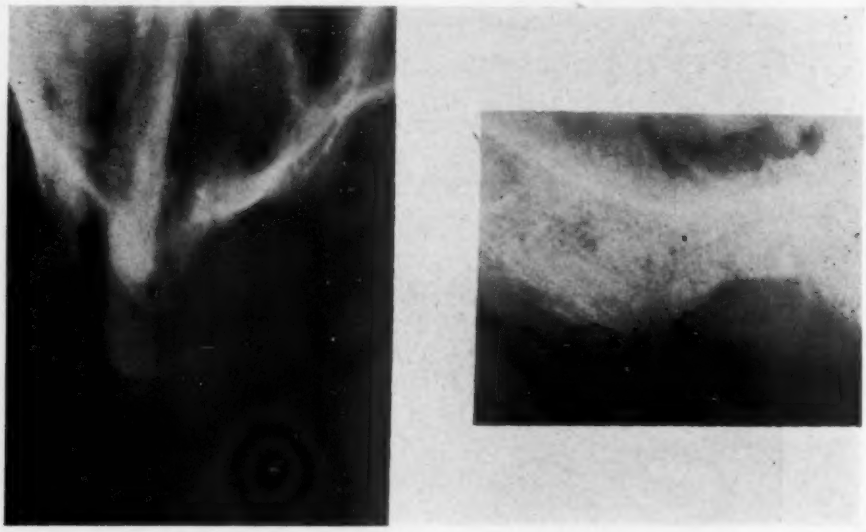

Fig. 4.—Giant cell tumor of maxilla. Note soft tissue tumor mass and type of bone destruction.

CASE 5.—L. H., male, aged 42 years. The patient noted a slowly growing mass in the lower anterior jaw for about three years. It had not caused him any discomfort, and he sought consultation because his friends were calling his attention to it.

Examination.—A nodular mass about the size of a small filbert was revealed. In several places the surface epithelium was denuded. It was not painful to touch, and neither did it bleed. The mass extended over the anterior mandibular ridge between the right lateral and left central incisors (Fig. 5).

X-Ray Examination.—X-rays revealed a superficial bone loss (Fig. 6).

Operation.—The tumor was removed en masse together with the involved bone. The bony cavity was cauterized.

Microscopic Diagnosis.—Peripheral giant cell tumor.

CASE 6.—D. B., female, aged 29 years. Nine months previously, the patient had noticed a small growth between the maxillary right cuspid and first premolar. It had progressively increased in size, covering the teeth in places and extending palatally to the mesial aspect of the right central incisor, and buccally to the distal aspect of the first molar. The cuspid and first premolar were separated by part of the tumor (Fig. 7).

Fig. 5.—Peripheral giant cell tumor.

Fig. 6.—Peripheral giant cell tumor with bone involvement. Note the gradation in the bone density and beginning resorption of mesial surface of lateral root.

X-Ray Examination.—X-rays disclosed slightly more than superficial bone destruction. A marked feature of the process was the clean-cut punched out resorbed areas in the roots of the teeth which were imbedded in the tumor (Fig. 8).

Operation.—The teeth, tumor, and bone were removed en masse and the area thoroughly cauterized. A plastic flap operation was performed to cover the area of excision.

Microscopic Report.—The specimen appears to be a section of maxillary bone, the gingiva in relationship to it, and four teeth imbedded in the specimen. The specimen measures 3 cm. in length and the teeth are seen to be two premolars and one right central incisor, and what appears to be an adjacent lateral incisor. Between the lateral incisor and the first premolar there is a

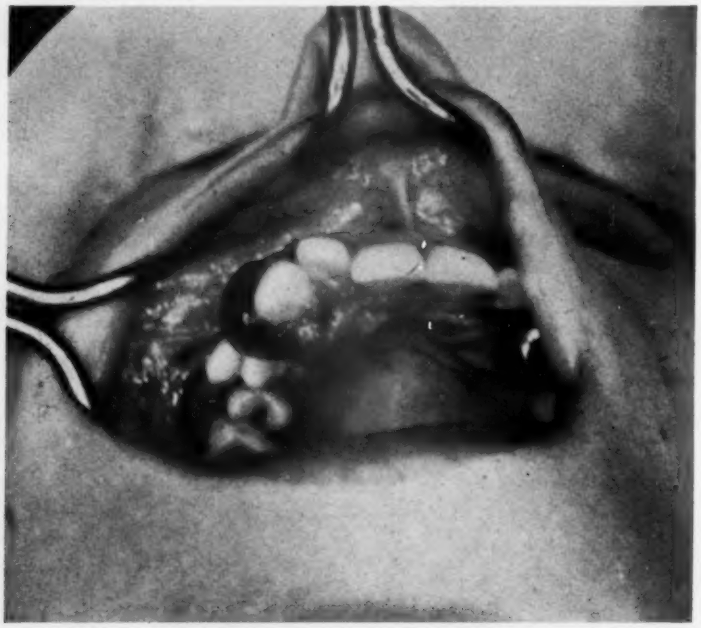

Fig. 7.—Peripheral benign giant cell tumor of maxilla.

Fig. 8.—Note thinning and resorption of roots involved in giant cell tumor.

space measuring 0.8 cm. in length, and the gingiva in this region is slightly protuberant and swollen, being covered, however, by unbroken mucosa, and is of a grayish-lavendar and grayish-blue color. The aspect of the specimen directly opposite that portion in which the teeth are imbedded contains several bone spicules, is a reddish-gray color, and moderately bloodstained. On the aspect of the gingiva behind the teeth previously described, the swollen portion of the gingiva is more pronounced, more easily recognized, and here the tumor mass measures 1.5 cm. in length. Upon cutting through this tumor mass, the mucosa of the gingiva is found to be unbroken over it, and the tumor itself

is found to consist of reddish-tan and yellowish-pink, together with a silvery-gray, slightly bloodstained, gelatinous appearing tissue which extends upward for a variable distance and at one point appears to have invaded the portion

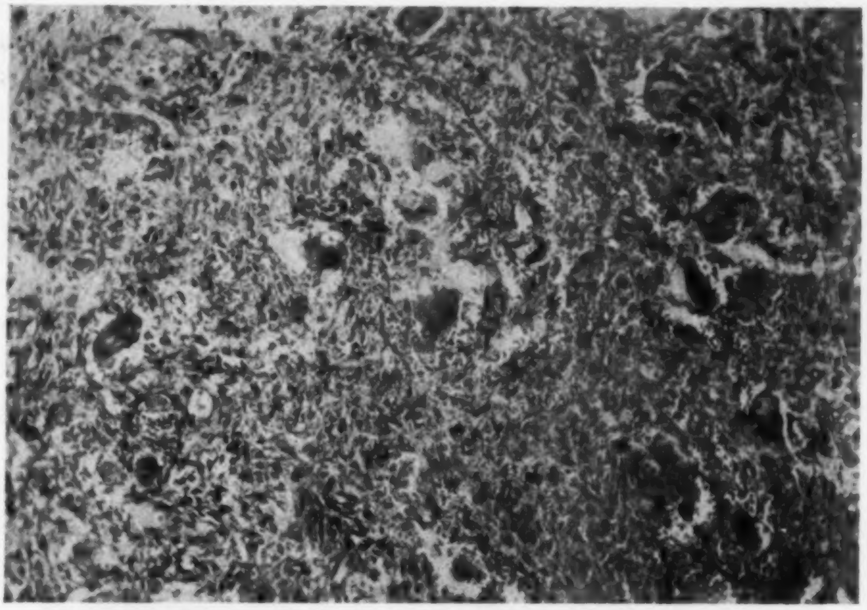

Fig. 9.—Low-power magnification. Peripheral benign giant cell tumor in Case 6. Tissue is composed of fibrillary cells, small round cells and multinucleated giant cells.

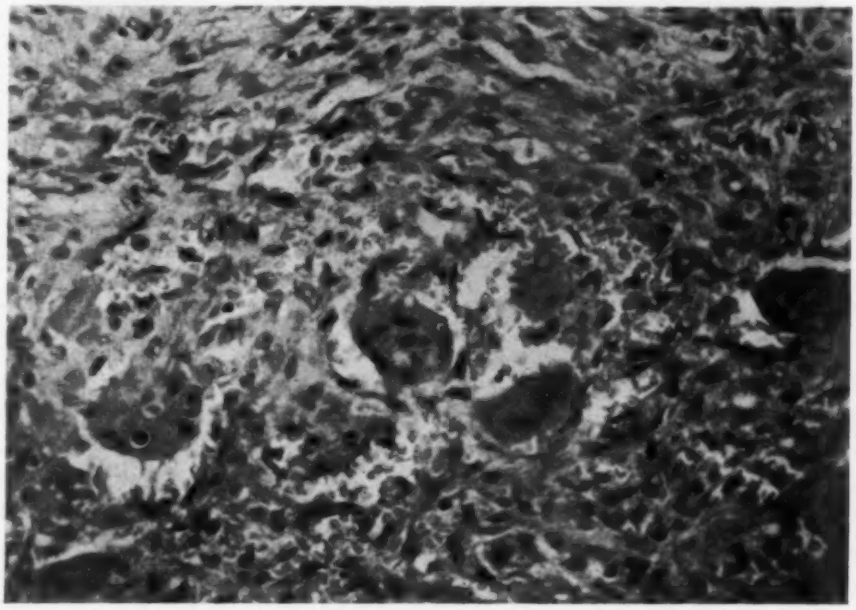

Fig. 10.—High-power magnification. The nuclei in the giant cells range in number from about seven to thirty.

of the maxillary bone in this region. Upon dissecting the tumor mass away from the roots of the teeth, the root of the right lateral incisor previously mentioned is found to be thinned out. Its ordinarily smooth surface is granular, roughened and irregular, and into this thin, roughened, and irregular area are

extensions of somewhat gelatinous appearing tissue from the main body of the tumor mass.

The section is covered over one edge by typical stratified squamous epithelium, which send well-marked rete pegs down into the connective tissue beneath it. Within this connective tissue, beneath the stratified squamous epithelium there are numerous small endothelial-lined capillary spaces, and the stroma is very rich in what appears to be young fibroblasts, with large, oval, relatively clear nuclei containing scattered basophilic granules. Also scattered throughout this connective tissue stroma, beneath the stratified squamous epithelium are areas heavily infiltrated with round cells, both small and large; and scattered throughout these infiltrated areas are collections of golden-yellow, granular pigment, of varying size, some being diffusely scattered, others being gathered into clumps. Deeper within the connective tissue, the

Fig. 11.—Peripheral benign giant cell tumor of mandible.

stroma appears less cellular and more densely fibrillary and contains occasional scattered groups of large and small round cells and scattered deposits of golden-yellow pigment similar to that just described in the more superficial portions of the section. A striking feature within the deeper portions of the section is the presence of moderate numbers of multinucleated giant cells, a few of which are small, containing on the average seven to twelve nuclei, but most others being large in size, with relatively clear cytoplasm and containing great numbers of nuclei, varying from ten or fourteen to twenty or thirty in number (Figs. 9 and 10).

Microscopic Diagnosis.—Fibrosing peripheral benign giant cell tumor.

Comment.—The thin punched-out areas in the roots, no doubt, were the action of the giant cells in the tumor. This gave rise to the possibility of the tumor being an osteoclastoma. However, blood studies were made and

x-rays of the long bones of the body taken. There was no increase in serum calcium or decrease in serum phosphorus. The phosphatase was within normal limits.

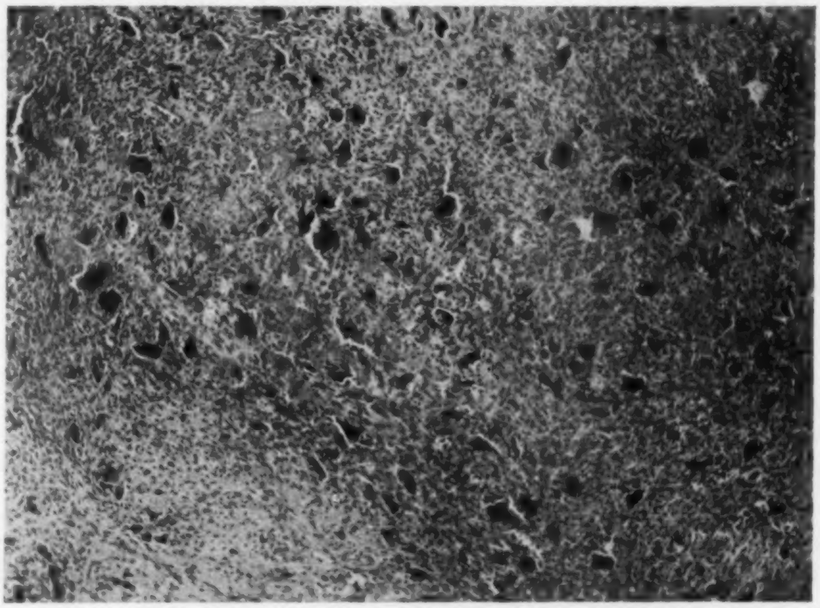

Fig. 12.—Low-power magnification. Peripheral benign giant cell tumor of mandible (Case 7). Tissue composed of spindle-shaped, round cells and multinucleated giant cells.

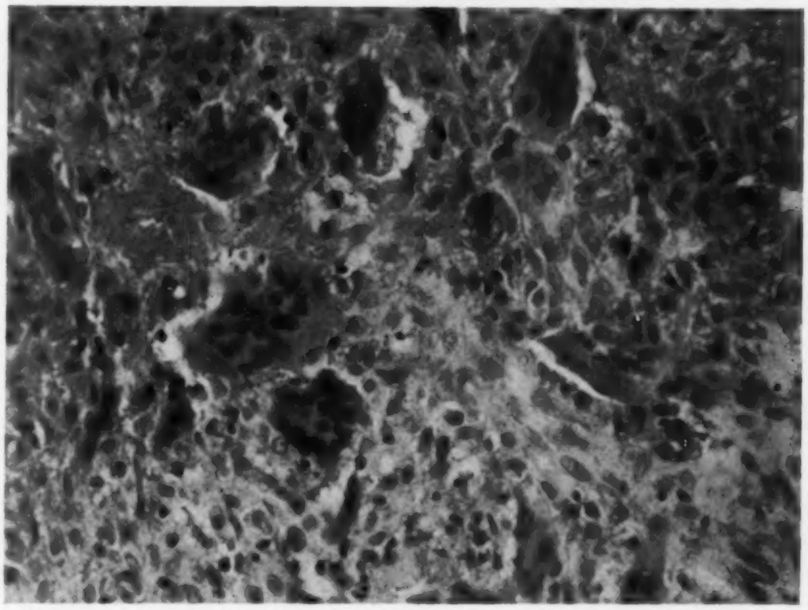

Fig. 13.—High-power magnification (Case 7).

The blood examination was as follows: Hemoglobin 75 per cent, red blood cells 4,830,000, white blood cells 13,250, 63 per cent polymorphs, 26 per cent lymphocytes, 10 per cent monocytes, and 1 per cent eosinophiles. No pathology in the other bones was found.

Case 7.—J. L., male, aged 47 years. The patient had noticed a growth in the lower right jaw for about three years. In the last year, it had increased in size very rapidly.

Examination.—Examination disclosed a tumor mass on the ridge of the right mandible in the second and third molar areas. It was bluish in the center, fading out to a pink color at the periphery. It did not bleed when manipulated (Fig. 11).

X-Ray Examination.—X-rays showed a superficial bone loss.

Operation.—The tumor was excised and the area cauterized.

Microscopic Examination.—The tumor was made up of round cells, fibrillary and spindle-shaped cells, and many multinucleated giant cells (Figs. 12 and 13).

Microscopic Diagnosis.—Peripheral giant cell tumor.

SUMMARY

There are two types of central benign giant cell tumors which must be differentiated. Some are osteoclastomas and part of the picture of hyperparathyroidism, and others are solitary benign tumors.

The distinguishing characteristics of the giant cell tumor are the multinucleated giant cells, absence of encapsulation and the property of bone destruction by foreign body reaction.

In all cases, a blood study for hyperparathyroidism, and x-rays of pelvis, long bones, and skull should be made.

The peripheral giant cell tumor may be differentiated from other epulides by color, texture, and type of bone loss. Accurate diagnosis can be made only by microscopic examination.

311 Commonwealth Avenue

A TEN-YEAR STUDY OF A CASE OF OSTEOFIBROSIS

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In March, 1937, I was privileged to read a paper entitled "X-Ray Changes Following Treatment of Osteofibrosis," before the International Association for Dental Research, in Baltimore. Since that time, I have carefully checked the case reported with roentgenographic series, and have found some interesting results. The following is a brief summary of the case:

The patient, a Cuban, aged 37 years, a cigar maker by occupation, presented himself at the New York University College of Dentistry for periodontal treatment in January, 1934 (Fig. 1).

Fig. 1.—Cuban, male, aged 37 years. Shallow periodontal pockets, excellent color of teeth, slight to moderate mobility of teeth.

Medical Report.—All tests were negative, except for the following symptoms: rapid pulse, increased blood pressure (169/98), tremors of fingers, slight prominence of eyes, slight enlargement of thyroid gland, and nervousness. The basal metabolism test was +30. (At subsequent tests, there was so much variation in the basal metabolic rate that it was believed to be unrelated to the mouth condition.)

Oral Examination.—1. Clinical examination indicated a generalized suppurative periodontoclasia with moderate mobility of the upper and lower anterior teeth and all the molars.

2. All the teeth responded to normal vitality tests, with the exception of left second and right third molars.

3. Traumatic occlusion was present and was exaggerated by an occupational habit of the patient, namely, the biting-off of cigars with the anterior teeth in the process of making them. (Since 1934, the patient has been a Pullman porter.)

4. Roentgenographic examination revealed extensive bone resorption throughout the entire mouth and unusual rarefaction about the apices of the

Delivered before the New York Section of the International Association for Dental Research, Jan. 27, 1944.

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lower teeth from left first premolar to the cuspid tooth on the right side. These conditions were also noted on the upper left lateral incisor and lower left second molar teeth (Fig. 2).

An opinion of this condition was secured from the late Dr. Abraham L. Greenfield, and his report follows:

"From the roentgenographic standpoint, it is utterly impossible to make a differential diagnosis between osteofibrosis and a granuloma. Clinically, the differential diagnosis can be made. If the roentgenograph reveals a radiolucent area about the apex of the tooth, and if the vitality response is positive and the color of the tooth is normal, we can make a fairly conclusive diagnosis of osteofibrosis.

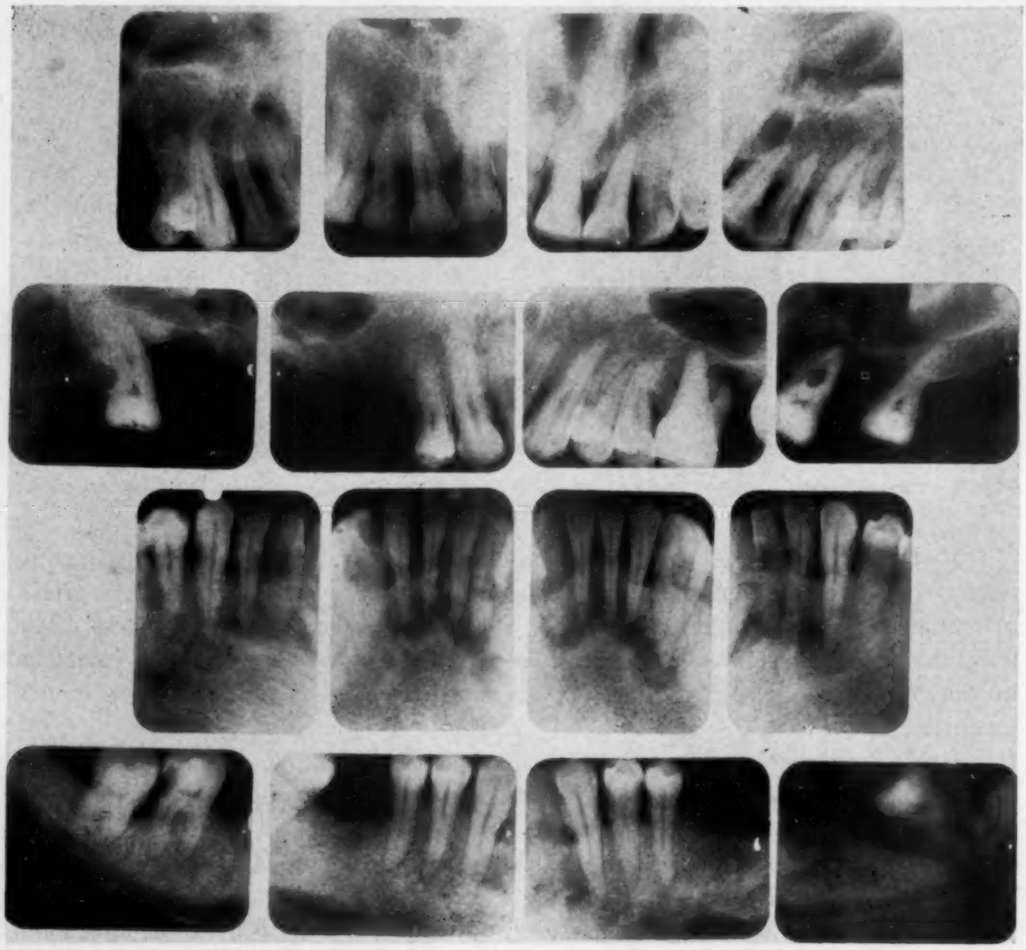

Fig. 2.—Roentgenographic series of case. Note rarefied areas about the apices of mandibular teeth and about the upper left lateral incisor (January, 1934).

"The radiolucent areas seen in the roentgenographic series are due to a fibrous degeneration which the bone undergoes. The diagnosis is osteofibrosis which indicates a replacement of bone by fibrous tissue."

Pathologic Report.—A biopsy was made of the lower right cuspid. The tooth and surrounding tissues were removed and a section was made of the

apical area. The following report was rendered by the pathological laboratories of Dr. Charles G. Darlington, Professor of Dental Pathology:

Gross examination shows several small pieces of bone. For microscopic examination sections of the specimen were made up of small irregular pieces of viable bone and adherent tissue. The bone varies in density from jagged

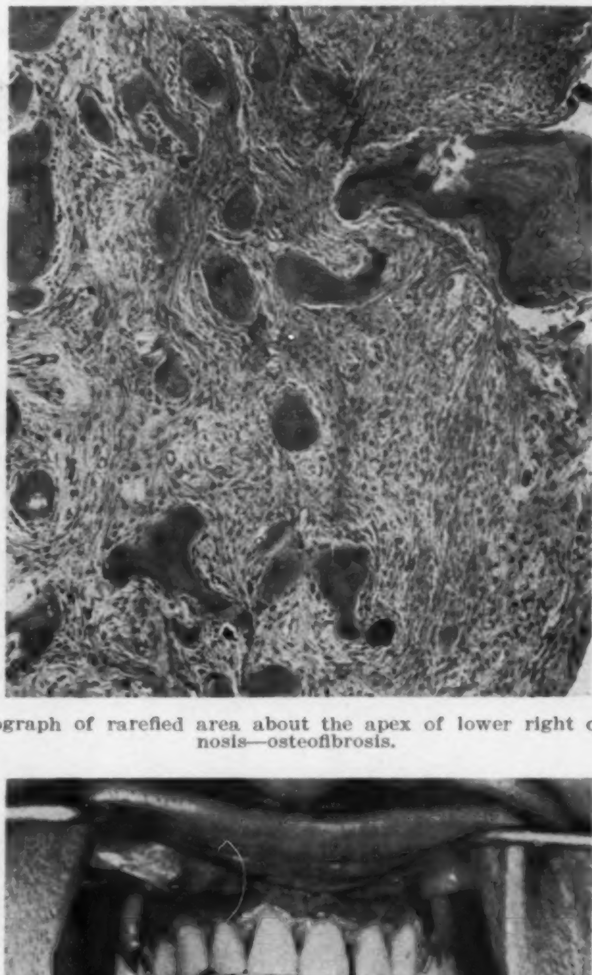

Fig. 3.—Photomicrograph of rarefied area about the apex of lower right cuspid tooth. Diagnosis—osteofibrosis.

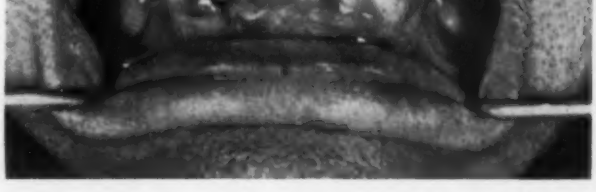

Fig. 4.—Partial denture which was constructed during periodontal treatment. At this time, equilibration of occlusion was completed (September, 1934).

plates to irregularly conjoined bone trabeculae. The interspaces, i.e., marrow spaces, vary in size and shape with the density of the bone. Where the bone is laid down in platelike masses, the marrow spaces show a light, delicate, poorly cellular connective tissue. Here the "marrow" is quiescent. In other

fields, where the bone is jaggedly trabeculated, the intervening spaces show a dense, richly cellular fibroblastic tissue. Here the "marrow" is active and one finds an occasional bone giant cell. Occasionally, in these fields, purple staining pools of lime salts impregnate the tissue. These pools are poor in cells and while of probable bony origin simulate cementum rather than actual bone. The tissue which adheres to many of the bone spicules is a tough dense connective tissue. The diagnosis is osteofibrosis (Fig. 3).

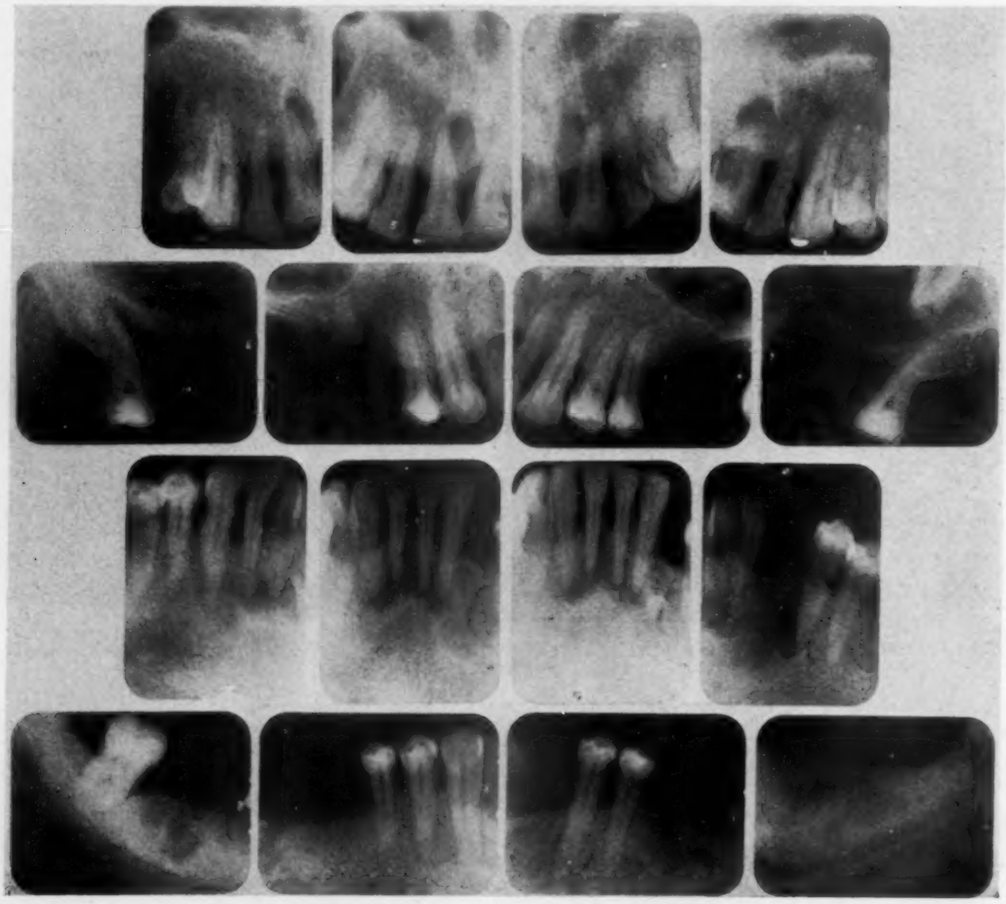

Fig. 5.—Note improvement in the regions of osteofibrosis. However, new areas developed in the region of the right premolars where none had appeared previously (March, 1935).

Treatment.—The etiology in this case was rather obscure and the following procedure was instituted.

1. Since the basal metabolism report was believed to be unrelated to the mouth condition, no further attention was given to it.

2. The upper right first molar, lower left second molar, lower right cuspid, and right molar teeth were extracted.

3. The occlusion and occupational habits were studied to determine whether these factors might cause a loss of lime salts in the areas indicated by the x-rays.

4. The periodontal condition was treated; it responded favorably. The treatment instituted consisted of balancing of the occlusion, instrumentation, toothbrush massage, and instruction in diet.

5. Partial dentures were constructed in September, 1934, which relieved the strain on the anterior teeth, and distributed the stresses on the teeth in all excursions of the mandible (Fig. 4).

It was hoped that the removal of the abnormal pressure on the teeth would tend towards a redeposition of lime salts in the areas from which it had disappeared. Vitality tests in March, 1935, indicated normal vitality responses.

Roentgenographic series, taken in March, 1935, six months after construction of dentures, indicated that there was noticeable improvement in the regions of the upper left lateral incisor, lower anterior, lower left cuspid and premolar. However, new areas developed in the region of the lower right second premolar. This occurrence in an area where the lesion had not appeared previously led us to believe that it was due to the overloading on these teeth which had been clasped. We must note, too, that the lower right molar had been extracted and that a posterior "stop" was missing (Fig. 5).

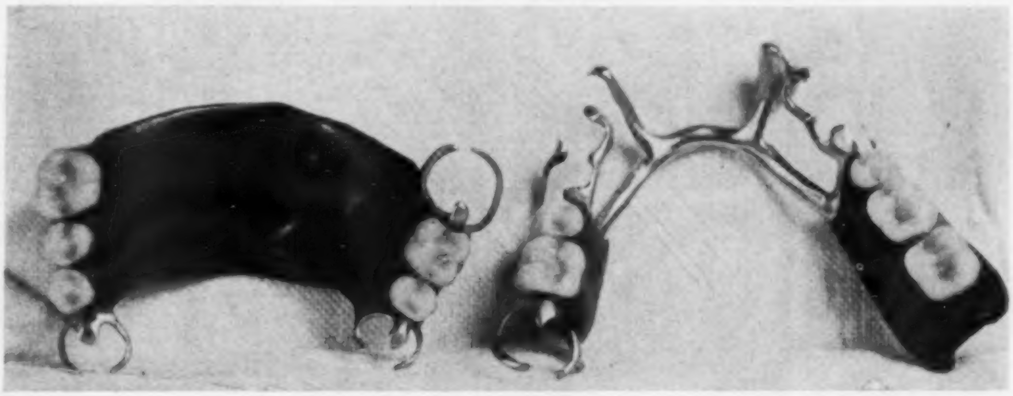

Fig. 6.—Photograph of the dentures. Note how reciprocal anchorage was obtained.

Fig. 7.—The new denture in position. Note lug on the lower cuspid. A space for the lug was ground into the enamel previous to the construction of the new lower denture (April, 1935).

A new denture was constructed (Fig. 6). This denture utilized more teeth for greater reciprocal anchorage (Fig. 7). Roentgenographic series taken in September, 1935 (one year after construction of first denture), shows great improvement, particularly in the areas around the lower right premolar teeth.

Subsequently, it was found necessary to remove the upper left molar, which had only been retained to give the denture better support. The upper denture was repaired, and in January, 1937, two and a half years after the first denture was constructed, a new series of roentgenograms was taken, which showed definite improvement. A physical examination at this time indicated a healthy individual, with a basal metabolism rate of -12, blood sugar 102, and blood pressure 142/90.

Fig. 8.—The patient did not return for checkup until January, 1944, exactly ten years after the beginning of treatment. This series indicates the normal appearance of the alveolar structures which had undergone severe changes ten years previously. The upper left lateral incisor was being traumatized by the lug on the lower cuspid, and treatment is now being instituted to bring the tooth into better alignment and to relieve the trauma (January, 1944).

Since the publication of the paper in 1937, another careful checkup was made when the patient was available, and I am happy to report that our hopes in 1937 were completely realized. All the rarefied areas have filled in with normal bone, despite the fact that the patient did not return as frequently as advised, to check on his dentures.

The complete roentgenographic series from 1934 to 1944 clearly demonstrate the excellent results which had been obtained. The set taken in 1944 is shown in Fig. 8.

The author is greatly indebted to Drs. Darlington, Schuyler, Swenson, Winter, Miller, Greenfield, Salman, Wald, and Troppozano, of the New York University College of Dentistry, for their cooperation.

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HERPETIC STOMATITIS: A REPORT OF SIX CASES OCCURRING IN ONE FAMILY

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IN RECENT years the dental profession has placed more and more emphasis upon the diagnosis of diseases of the mouth. While formerly most oral conditions were classified either as "Vincent's infection" or "Aphthous stomatitis," bacteriologic studies and more astute clinical observation have established the existence of definite oral diseases formerly classified in these two large groupings. The vast amount of clinical investigation on the oral manifestations of avitaminoses has stimulated the clinical phase of this differentiation. Recent work, during the past five years, has definitely established the existence of herpetic stomatitis as a separate disease entity.

W. C. Black[1] separated this condition from Vincent's infection by clinical examination, but also suggested a virus as the etiological agent. Dodd,[2] and his co-workers, established the herpes virus as the etiological agent by inoculation of the material from the oral lesions into a rabbit's cornea. The reaction obtained was the same as with the herpes virus. They not only established a definite etiology for the clinical syndrome, but provided a convenient laboratory test for the corroboration of the diagnosis.

The contagiousness of this disease was indicated by Scott, Steigman, and Convey[3] who reported a history of contact in about 50 per cent of their cases. Levine, and his co-workers,[4] have described a vesicular stomatitis, which they believed to be of herpetic origin. It occurred as an epidemic near a lake in New Hampshire with 106 persons affected. The disease was characterized by a high infectivity, temperature of 104° F., and the presence of vesicular lesions which subsequently ulcerated. These may be found in the throat, and sometimes in the oral cavity, the buccal mucosa, and the gingival margin (Thoma[5]).

Ziskin and Holden[6] have described the bacteriology, immunology, and clinical appearance of the disease. In describing the condition they state that frequently, the first sign of the infection is a herpetic lesion on the lips. Vesicles may appear anywhere on the keratinized gingivae or buccal mucous membranes, or in the throat. Vesicles may appear on either the skin or mucous membranes. The free gingival margins and the interdental papillae are also often involved. The alveolar gingivae are spongy and swollen crownward, the condition tending to bury the teeth. The regional lymph nodes are invariably swollen and tender. They found rectal temperatures ranging from 99° to 105° F. with an average of about 101° F. In their series of cases (twenty-one) the duration was from one to three weeks. Over half of their cases gave positive evidence of a history of contact (herpetic sores on lips), which is ample evidence of the contagious nature of the disease.

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We have previously reported a case with marked clinical manifestations of herpetic stomatitis.[7] The photographs presented therein illustrate clearly the type of lesions found in severe cases of this type. The following report deals with a series of six cases of herpetic stomatitis occurring in a family of eight children, illustrating the extremely contagious nature of herpetic lesions.

CASE REPORT

A Negro girl, 13 years of age, came to the hospital complaining of soreness of the mouth and a severe "cold." Six days previously she had had her first menstruation, accompanied by pain in the stomach and back. This had lasted for four days. During this time (according to her mother) the patient had had a severe cold accompanied by a "cold sore." The "cold sore" appeared every time the child had a bad cold. The "cold sore" was present for three to four days with soreness of the mouth for three days. After menstruation had ceased, the stomach and back pain disappeared, leaving persistent and continuous pain in the mouth and head.

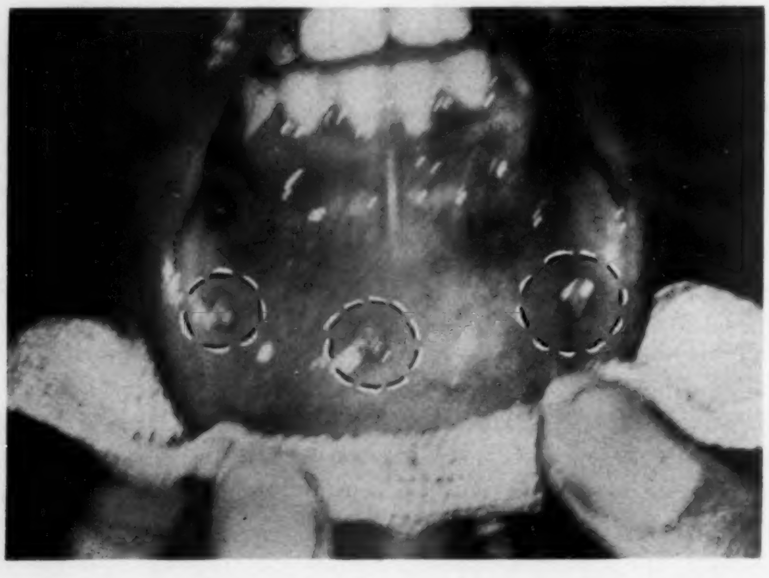

Fig. 1.

Examination of the patient revealed a slight bronchitis with a temperature of 101° F. A "cold sore" which may have been due to mechanical abrasion, was present on the upper lip, at its junction with the left ala nasi. The gingivae appeared red and inflamed, and in a slightly edematous condition. The red color was particularly marked at the gingival crests. Three small ulcers, yellowish in color, were present on the labial mucous membrane (Fig. 1). They were about the size of a large pinhead, slightly raised, with a red border, and were extremely painful to touch. The tongue and rest of the oral mucosa were not involved. The left submaxillary node was markedly palpable.

From the clinical appearance and history, a tentative diagnosis of herpetic stomatitis was made. To confirm this diagnosis, we decided to perform the "rabbit-eye test" of Dodd, as described by Ziskin and Holden.[6] An albino rabbit (male) was placed in a box so constructed as to leave only the head

visible. Two drops of a 1 per cent solution of holocaine were placed in each eye of the rabbit. A sterile 20 gauge hypodermic needle was then used to scarify the cornea of both eyes. Three scratches were made in each direction, making a criss-cross pattern. A sterile swab was then placed in the patient's

Fig. 2.—Control.

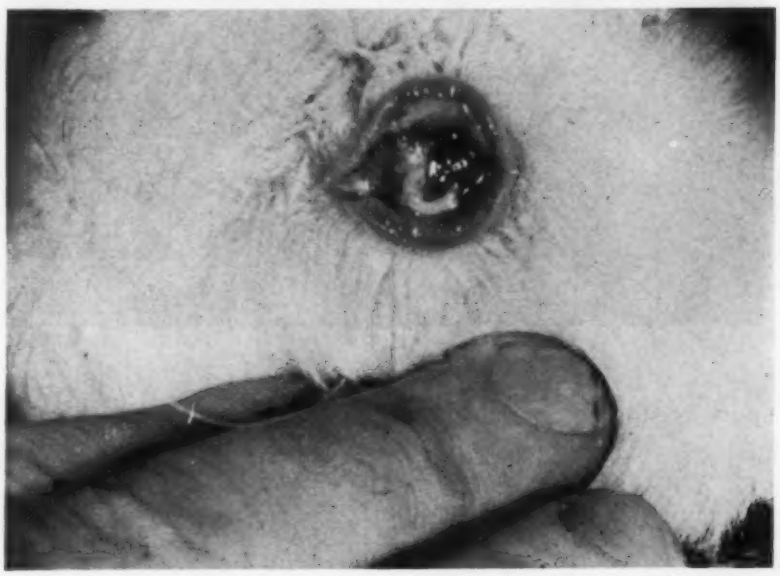

Fig. 3.—Test.

mouth, on the gingivae and in the saliva. This was applied to the left eye, the right eye remaining as a control. Examination, after 48 hours, showed the right eye to be completely normal. A very small ulcer appeared on the left one but no conclusions were drawn, since there was some disagreement as to whether this was a true "ulcer" due to the inoculation. After 96 hours, however, there

was no doubt as to a positive reaction. While the right eye (control) appeared completely normal (Fig. 2), the left eye (test) was almost completely closed, exhibiting marked photophobia, and exuding a purulent, serous exudate. The conjunctiva was markedly injected, the nictitating membrane was drawn half way across the eye, and the fur around the eye was matted from the exudate (Fig. 3). This then was the "purulent keratoconjunctivitis" described by Ziskin and Holden.[6] It disappeared in a few days leaving a marked opacity of the cornea. The diagnosis of herpetic stomatitis was then accepted.

The patient's mouth was painted with 1 per cent gentian violent solution. Hydrogen peroxide rinses were advised three times a day, accompanied by a bland liquid diet, reinforced with dilute fruit juices. The mother was warned of the infectious nature of the condition and told to "isolate" the child as well as she could. Although she had eight children, of which the patient was the eldest, and was near term with the ninth, she appeared extremely cooperative in trying to keep the child in bed and away from the others. All utensils, etc., used by the patient were to be kept separate.

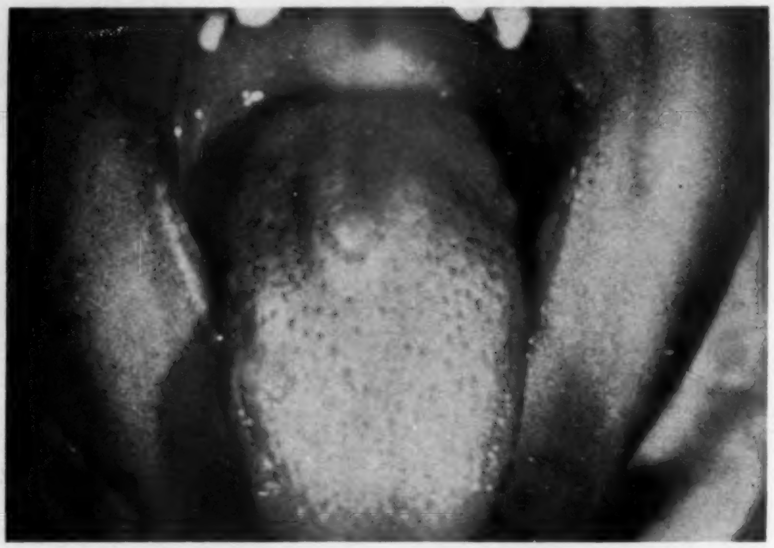

Fig. 4.

We had asked the patient to return in a few days. Because of the holidays and the mother's condition she failed to present herself. A card was sent to the mother, who replied that six of her eight children had contracted the disease. An appointment was immediately made, and the children were brought in for examination.

The ages of the children ran from 1½ to 13 years. The latter was the age of the original patient. The condition had spread during the four days following the onset in the first child. It varied in intensity from a sore mouth with inflamed gingivae seen in three children, to a full-blown stomatitis with herpetic ulcers and a temperature of 102° to 104° F. in two children. The severity varied with the ages. The two oldest children (13 and 11½ years) had the most severe cases, with ulcerations, fever, and a prolonged course of the disease. The

duration, ran from three days to almost three weeks, and seemed to vary in proportion to the severity of the condition. The presence of "cold sores" on the lip (herpes labialis) and on the nose was noted in three of the children. One child (5 years old) presented lesions on the dorsum and side of the tongue, eight days after the onset (Fig. 4). The lesions were about the size of a small pea, irregular in outline, slightly raised, with a red border, and surrounded by a small area of atrophied filiform papillae. They were of a faint yellow color with a large irregular light red center. One lesion seemed to have resulted from the coalescence of two individual "sores." One child who did not experience great pain or fever had a labial lesion at the angle of the mouth, which was still present after the oral symptoms had subsided.

In all patients, hypertrophy of the gingivae, with reddening of the gingival crests, was noted. Three presented mouth ulcerations as well. In two, occasional bleeding of the gingivae was still present after the herpetic condition had subsided. Only two of the eight children in the family had no signs or symptoms whatsoever and seemed entirely normal. It was presumed that they had some sort of immunity.

COMMENT

Because of the chain of events and the clinical symptoms, we concluded that the initial herpetic stomatitis had spread to the other children in spite of all precautions. The treatment prescribed for the first child was prescribed for all to ward off a possible complicating Vincent's infection, since no accepted treatment has yet been found for herpetic stomatitis. In addition, the mother was advised to raise the vitamin B and C intake of each child, using synthetic preparations if necessary.

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209 EAST 23RD STREET

SIMPLIFIED METHOD OF ROTATING SKIN AND MUCOUS MEMBRANE FLAPS FOR COMPLETE RECONSTRUCTION OF THE LOWER LIP

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DEFECTS of the lower lip result from inflammatory or degenerative processes, as advanced carcinoma[1, 2] and extensive ulcerations (noma); or traumatisms from burns[3] or other accidents.[4, 5] That reconstruction of these defects has been a surgical problem for many years is evidenced by the fact that according to Pierce and O'Connor,[1] over sixty-five methods and modifications of repairing complete or partial defects of the lower lip have been described in the literature. According to Martin,[6] von Bruns,[7] as early as 1859, had found thirty-two methods described by fifty-two authors. That there have been so many different methods for the correction of this defect is indicative of the inexpedience of any one method. This fact should decry, on the one hand, the advancement of a new technique which may be only as good as others previously advocated, and yet, on the other hand, should be a stimulant to the development of a technique which fulfills satisfactorily all surgical requirements.

Cheiloplasty for carcinoma of the lower lip, one of the oldest operations known, has been practiced since the days of Celsus (born about A.D. 25).[6] He is generally considered the originator of a method, the principle of which is still adhered to today—V-shaped incision and modifications including horizontal incisions from the angles of the mouth and along the lower edge of the mandible forming two lateral flaps of the cheek. According to Foman,[8] the Hindus utilized rotating flaps from the adjacent tissues to rebuild a defect of the lip; this later became known as the Indian method. Tagliacozzi is accredited with the first description of a technique using flaps from the arm (1597).[9] Chopart,[10] in 1785, reported the use of advancing flaps from the neck and chin for this purpose. Most of the operations advocated today are modifications of procedures, the basic principles of which were recognized in the early nineteenth century.

Bernard[11] and Burow and Saemann,[12] in 1853, devised an operation in which full-thickness triangles were removed from the upper lip and discarded. The lower lip was then built by loosening and pulling the sides of the cheeks to the midline over the mandible. This procedure was modified by Steward,[13] in 1910, and Martin,[6] in 1932.

In 1877, Estlander[14] proposed a procedure which is one of the recognized methods of lip reconstruction today. A V-shaped incision is made. A similar defect only one-half the size is outlined at the outer border of the corresponding upper lip and excised completely, excepting the tissue where the coronary artery of the lip is located. The upper triangle is then turned down into the lower lip and sutured. In 1936, Padgett[15] modified this operation by bringing

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the outer part of the lower lip to the center to form the middle portion of the lip. The outer part is repaired from the upper lip flap. There have been innumerable modifications of the Estlander operation.

An extensive lower lip defect is usually reconstructed by some form of pedicle transplant, sliding,[16] rotating,[17] or advancing[10] These transplants may be rotated from the tissue above together with a portion of the upper lip, they may be rotated from tissues beneath and lateral to the chin upward to rest on a buttress of tissue, or they may be rotated from the tissue above and lateral to the upper lip with the pedicle attachment incorporating a portion of the upper lip. In most methods of rotation flaps the necessity of a lining for the pedicle is ignored and frequently the mucous membrane lining the lips is overlooked. It is not unusual for the surgeon attempting reconstruction to underestimate the amount of tissue necessary to obviate the possibility of subsequent tightness of the lips. Consequently, a foreshortened, noticeably tight lip, which permits drooling, invariably results.

Sliding flaps, commonly fashioned from the tissues of the cheek lateral to the defect, are usually outlined by parallel incisions from the defect backward to the region at the anterior border of the ramus of the mandible. These flaps are, of course, as wide as the defect of the lip. This method is objectionable because it produces a tight lip which often breaks down because of the great tension. It is not only cosmetically undesirable but also functionally unsatisfactory; it narrows the orifice of the mouth proportionately, and makes the insertion of artificial dentures, when necessary, impossible.

There are numerous varieties of advancement flaps.[1, 6 19, 19a] These might appear to be ideal from a cursory glance of the diagrams illustrating their formation. However, only casual study of these illustrations is necessary to realize that in most instances pedicle flaps cannot be fashioned and made to heal to conformity. Here, again, the common criticism is the resultant foreshortened lip and the failure to include the mucous membrane lining. Not only are there associated residual lateral defects following reconstruction in this way, but good functional result of the new lip is doubtful.

Occasionally, reconstruction of massive defects of the lower lip by the use of tube pedicle transplants from distant regions has been advocated.[18, 20, 21] Because the lip consists of two layers of skin with no intervening muscle, repair according to this method gives a flabby lip, which is not good cosmetically. Then, too, the color and texture of the facial skin is only simulated by skin from the face, the forehead, or the mastoid region. The texture and color of skin taken from distant areas is almost uniformly at variance with facial skin. This type of repair probably should be resorted to only in those instances in which facial tissue is not available.

The following technique embraces simplicity and a satisfactory cosmetic and good functional result. Repair is accomplished by a combination of advancement flaps of both skin and mucous membrane. First, a pattern of the defect of the lower lip is made (Fig. 1). This pattern is then bisected, as only one-half the defect is reconstructed from its respective side. From the proper half of the bisected pattern an area of skin lateral and slightly inferior to the defect is outlined, identical in size and shape to the respective half of the

bisected pattern. The pattern, anchored at its extreme lateral and upper border, is so formed that it may be swung downward and back to its normal position. After the pattern has been swung back and forth a sufficient number of times to insure the surgeon that the proposed flap is large enough to cover the defect, the margins of the pedicle are outlined with indelible ink or dye (Fig. 2). Following the pattern designed in this manner, the surgeon dissects the pedicle through skin, fat, and muscle, taking care not to penetrate the underlying mucosa. The flap is so designed that it permits the incorporation of fibers from the triangularis muscle, thus imparting animation to the reconstructed lip and reflecting minimal disturbance in innervation and muscle function. After the pedicle has been raised and shifted forward into the defect and found to be adequate, it is wrapped in moist saline gauze. The same pattern used for outlining this skin and muscle flap is then carried inside the mouth where the upper and outer point is held fixed again, and in the same manner an area of mucous membrane identical in size and shape is raised and brought forward into the defect to form the lining of the lip (Fig. 3). The

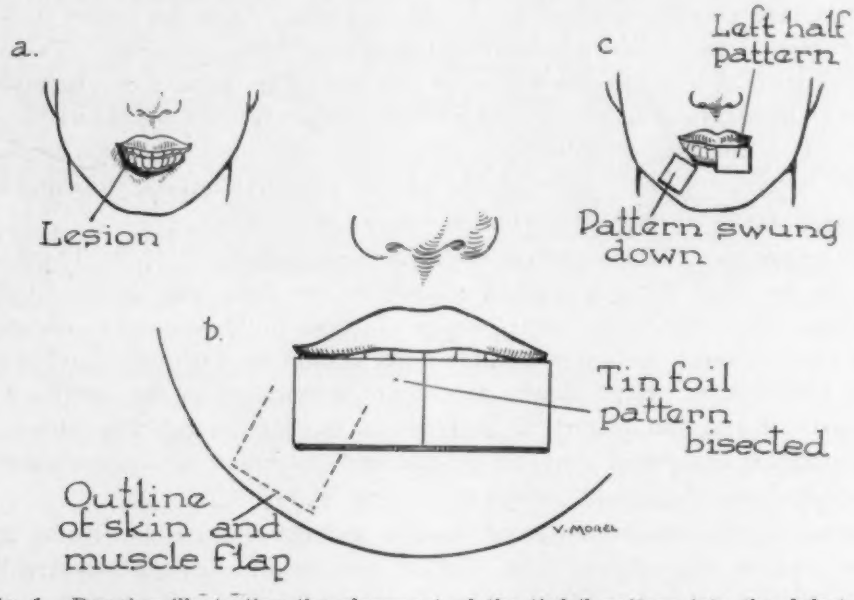


Fig. 1.—Drawing illustrating the placement of the tinfoil pattern into the defect, showing the location which the bisected half of the pattern assumes when it is shifted into its downward position preparatory to outlining the flap. Insert *a* shows the extent of defect and insert *c* shows the bisected pattern on the right swung down into position for outlining flap.

mucous membrane flap differs from the skin flap in only two essentials: it is not so thick and it is slightly wider. The purpose of this discrepancy in width is to enable the surgeon to bring the extra portion of mucous membrane over the upper border of the skin flap and to suture it to the outer margin of the flap in such a way that it forms a covering for the upper border of the skin flap. This provides the vermilion border of the lip. Great care is taken to avoid injury to the Stensen duct. Following the dissection of the mucous membrane flap and its rotation into its anterior forward position, the defect is closed by direct approximation (Fig. 4). If there is any apparent shortness in the length of the mucous membrane flap, it may be necessary to adjust the

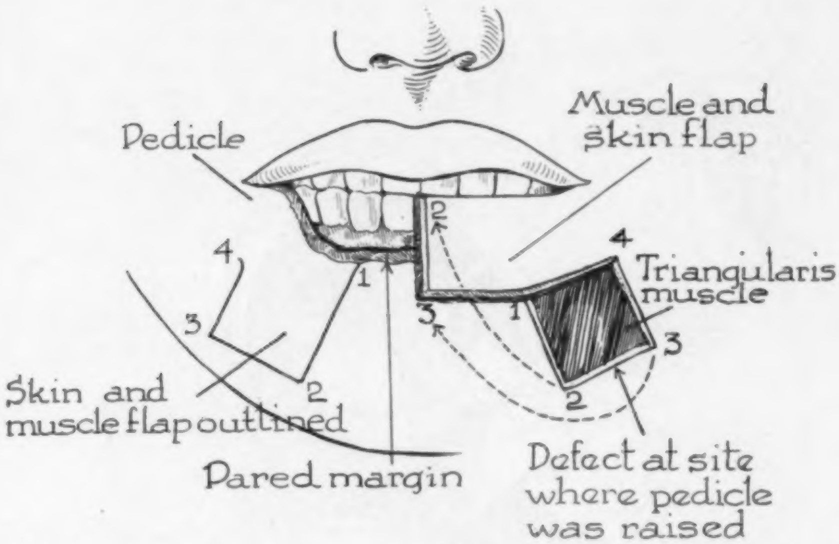


Fig. 2.—Drawing showing the flap outlined on the right and the flap dissected up on the left to include skin, fat, and a portion of the triangularis muscle. The flap on the left has been swung up into its proper position for its incorporation with the mucous membrane in reconstruction of the lip.

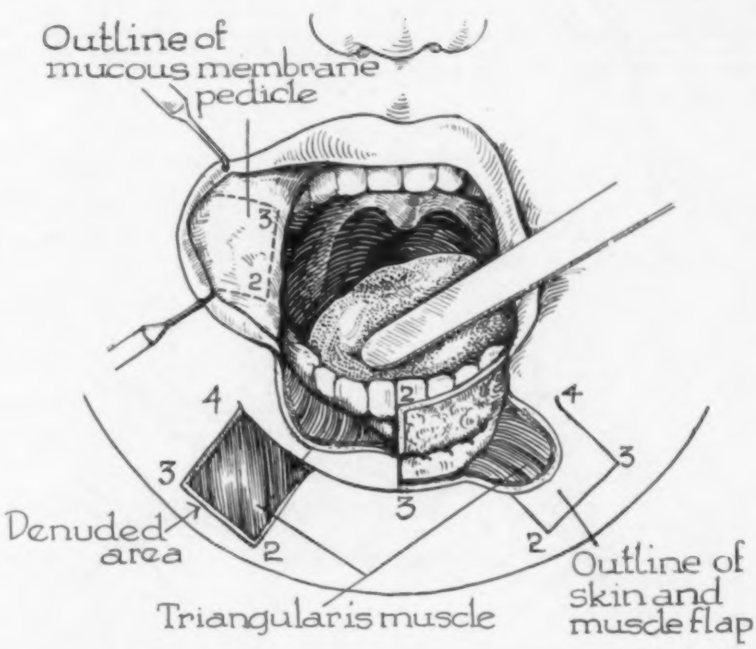


Fig. 3.—Drawing showing the outline of the mucous membrane flap over the buccal mucosa of the cheek on the right. The mucous membrane flap which was dissected up and advanced forward on the left is clearly shown. The skin and muscle flap on the right have been raised and brought into an upward position; that on the left is merely outline.

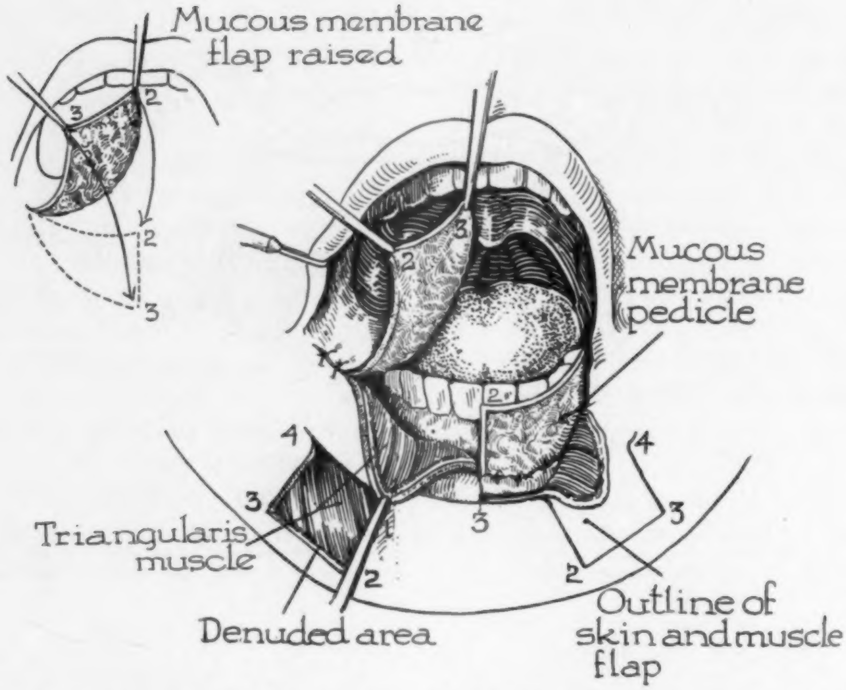


Fig. 4.—Drawing showing the advancement of the mucous membrane flap on the right cheek as it comes forward to join the skin and muscle flap. The mucous membrane flap on the left has already been advanced for the reception of the skin and muscle.

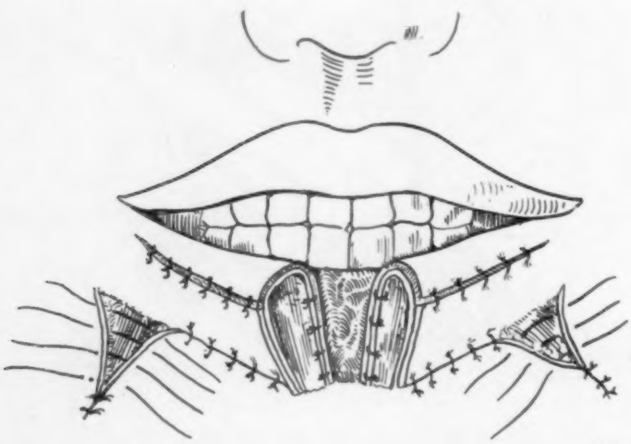


Fig. 5.—Drawing showing approximation of skin and muscle flap with the mucous membrane flap on either side. It also demonstrates the method of drawing the mucous membrane flap over the upper margin of the reconstructed lip where it forms the new vermilion border.

base of the pedicle. If the dissection has been carefully carried out and all measurements accurately followed, the mucous membrane flap will fall into apposition with the skin flap so that they adhere in a normal manner and the upper border of the skin flap will be adequately covered by the excess of mucosa (Fig. 5). This represents the reconstruction of one-half the lip defect. When necessary, for complete reconstruction of the lower lip, the identical procedure may be carried out on the opposite side.

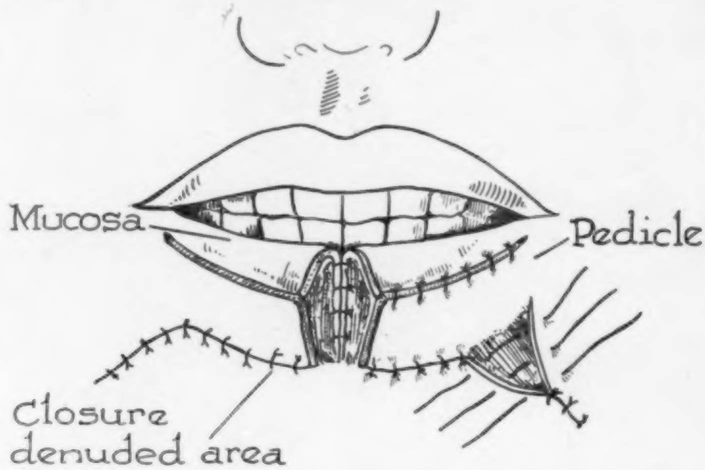


Fig. 6.—Drawing showing attachment of each composite flap with the other as they are joined in their final approximation to complete the newly formed lip.

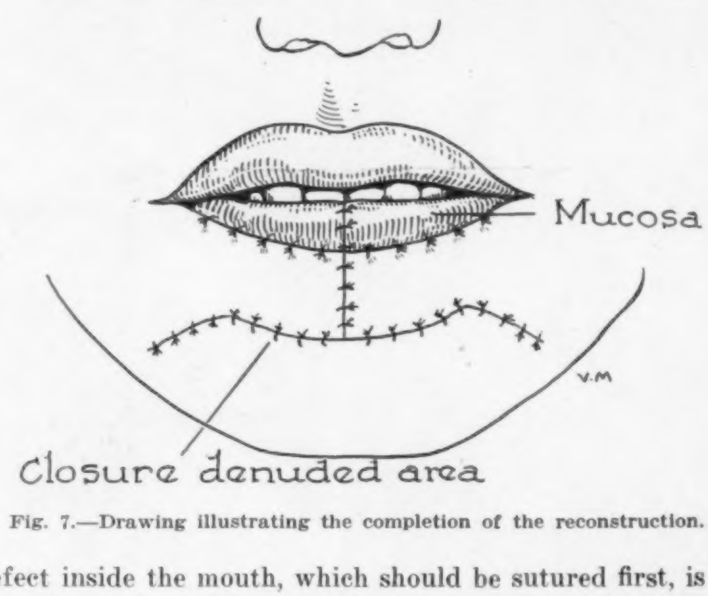
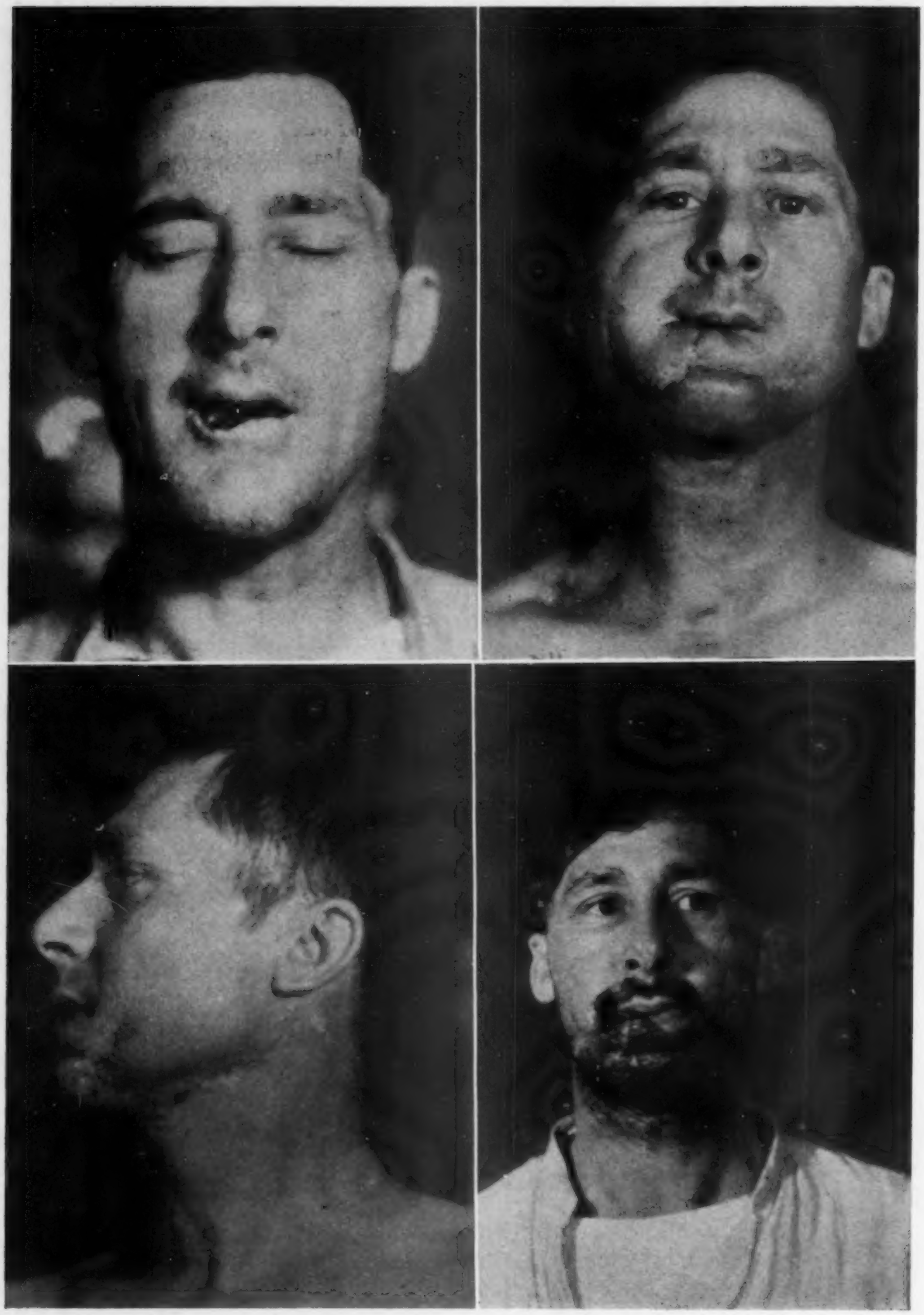


Fig. 7.—Drawing illustrating the completion of the reconstruction.

The defect inside the mouth, which should be sutured first, is closed with little or no difficulty by means of interrupted sutures of small black silk or 0000 silkworm gut. This line of closure continues along the entire inferior border of the newly formed mucous membrane flap (Fig. 4). The skin flap is then brought up into its position where it serves to reconstruct part of the defect of the lower lip, and the resulting defect lateral to the skin flap is closed by interrupted sutures of No. 1 black silk (Fig. 5). The flap usually will remain in place without tension. Approximation of the inferior margin of the

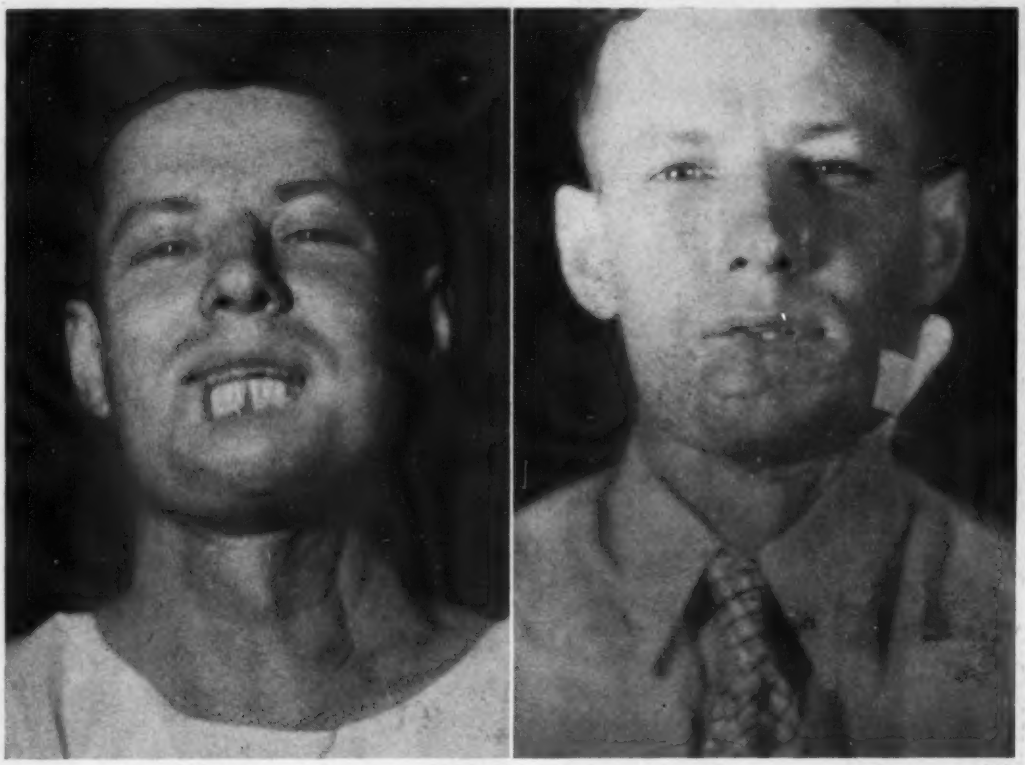
A. B.



C. D.

Fig. 8.—*A*, Photograph showing extensive carcinoma around the right half of the lower lip. *B*, Photograph taken ten days after operation. Procedure designed to remove all carcinoma and to reconstruct completely the lower lip at the same time. *C*, Lateral view taken ten days following operation. *D*, Photograph taken approximately six weeks following operation. Although the details of the lip are obscured by the beard, one can see that the lip has undergone considerable relaxation and the tissues are softer and under less tension at this time.

skin flap with the margin of the defect is then carried out with interrupted sutures of 0000 silkworm gut and an occasional suture of small black silk for tension. If this procedure is carried out accurately and meticulously there will be no tightness, shortness, or tension (Fig. 6). The excessive mucous membrane comes over the upper margin of the skin borders of the skin flap to form an entirely satisfactory vermilion border (Fig. 7). Two deep retention sutures of No. 50 white cotton are taken to hold the two skin flaps in close apposition in the midline. Other sutures of 0000 silkworm gut are added as interrupted ones. Because of the danger of infection in this type of wound,



A. B.

Fig. 9.—*A*, Photograph showing defect resulting from removal of squamous carcinoma of the lower lip. *B*, Photograph taken approximately six weeks after complete reconstruction of the defect of the lower lip.

a small rubber dam drain is inserted through a stab wound made in the skin below the reconstructed lip and carried up to the inferior margins of the new lip by blunt dissection with curved scissors. This is left in place for forty-eight hours. Because this type of wound is subject to excessive contamination and because of the possible development of Vincent's angina, except when contraindicated, a prophylactic dose of neoarsphenamine intravenously is routinely administered. An additional 5 per cent solution of neoarsphenamine in glycerin is applied three times daily along the suture line.

This procedure has been carried out on twelve patients. The cosmetic results have been uniformly good, the functional results gratifying in all

A.

B.

C.

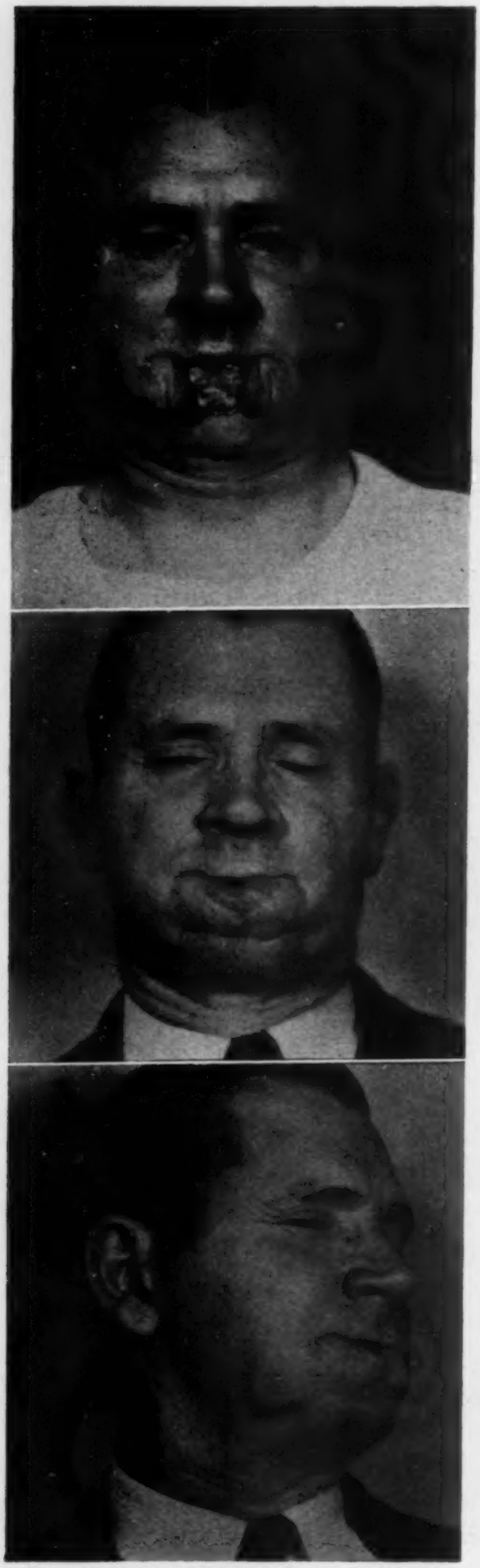


Fig. 10.

A.

B.

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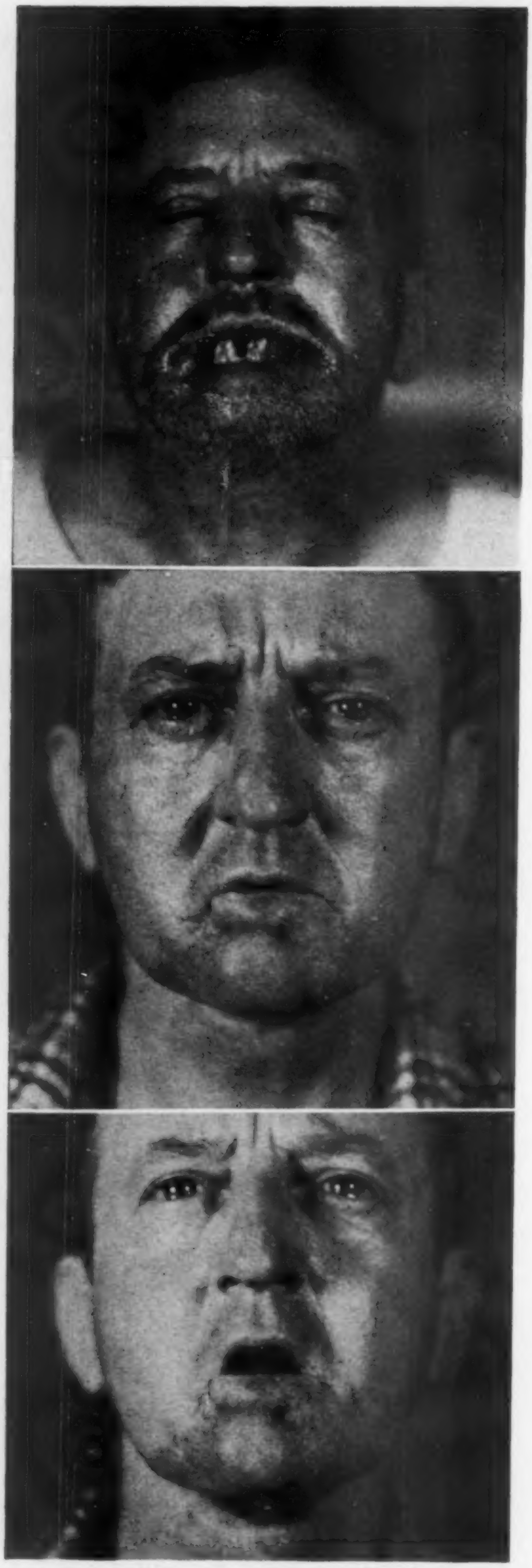

Fig. 11.

Fig. 10.—*A*, Defect of the lower lip resulting from extirpation of the carcinoma. *B*, Photograph taken approximately two months following reconstruction of defect of the lower lip. *C*, Lateral view of reconstructed lip taken approximately six weeks after time of reconstruction.

Fig. 11.—*A*, Defect of lower lip following extirpation of squamous cell carcinoma. *B*, Appearance of the lip approximately six months following reconstruction. *C*, View showing the ability of the patient to obtain good open bite following this reconstruction.

twelve cases. It is significant that in all of these patients there has been a noticeable absence of tightness or shortness of the lips and in no instance has a patient complained of drooling.

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Case Reports

CASE NO. 90

MANAGEMENT OF AN UPPER CUSPID IMPACTION

MATTHEW LOZIER, B.S., D.D.S.,* NEW YORK, N. Y.

THE principal reasons for reporting this case are the consideration of the factor of localization employed in the diagnosis and the conservative surgical approach resorted to in the removal of a deeply impacted canine tooth situated in close proximity to some of the other teeth in the jaw.

A girl, about 20 years old, in apparently good health, presented herself for diagnosis and treatment of an upper impacted right canine. The clinical examination revealed a full complement of teeth with a normal occlusion, with the upper right cuspid missing and with the corresponding temporary cuspid still in position. The radiographic examination disclosed a deeply impacted right upper canine lying diagonally, with its crown inclined downward over the roots of the adjoining central and lateral incisors. Though the tooth was entirely symptomless, its removal was deemed advisable because of the observed existing rarefaction around its crown, suggesting a follicular cyst, which would, it was thought, within time, affect and destroy the incisors.

Localization.—The problem of determining the location of the impacted tooth was not as simple as it is ordinarily. The bite-film approach, so often completely relied upon in locating impacted upper anterior teeth, has never been a favorite with me, in spite of frequently heard declarations as to its usefulness. The case on hand, as can be readily observed from the presented bite films obtained by overhead angulation, again conclusively showed the difficulty in determining the relative position of the impacted tooth to the alveolar plates as well as to the roots of the adjoining teeth, when this method of localization was used. As a matter of fact, if one is to attempt to decide upon the location of the impacted tooth from this radiograph alone, it could be readily assumed that the crown of the tooth lies here palatally while its root portion, which cannot be clearly discerned, is apparently situated more anteriorly, toward the labial aspect. However, when the Clark method of localization was employed (a technique based upon the principle that the more distant object from the observer moved in the same direction that the observer moves, and vice versa), it was decided that, in all probability, the reverse is true here. The complete soundness of this diagnosis was substantiated later through the surgical findings. It can also be added that there was absolutely no indication in the contour of the arch as to just where the tooth was situated, because the

*Formerly Instructor in Oral Surgery, New York Post-Graduate Medical College Hospital.

"movement" of the crown of the impacted tooth during the employment of the Clark method of localization, as evidenced by the radiographs, is very slight here. Impactions found on labial and buccal aspects of the upper maxilla are decidedly uncommon and, in this case, the preservation of the patient's incisors was greatly desired; therefore the approach to the removal of the tooth was undertaken with a certain degree of anxiety and hesitancy.

Fig. 1.

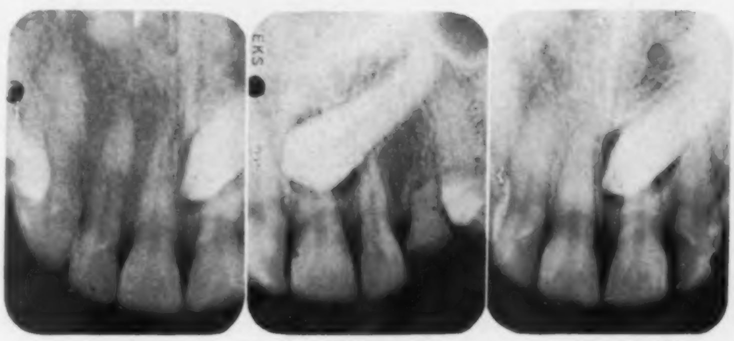

Fig. 2.

Surgical Procedure.—Before proceeding with anesthesia, all of the adjoining teeth, including the temporary cuspid, were tested for vitality and found entirely normal. A mucoperiosteal flap extending from the center of the crown of the left central to the distal of the crown of the right temporary cuspid was raised and the bone well exposed to view. A small narrow section of bone located exactly between the distal aspect of the right central and mesial aspect of the right lateral incisor was carefully removed and the neck of the impacted canine exposed to view. This exposed portion was then sectioned with an engine-driven surgical mallet. The crown, that is, half of the tooth, was then

moved a bit distally into the space created during the sectioning of the bone and tooth. With the aid of small curved instruments, it was dislocated out of its bed and the cystic membrane enucleated. The root portion of the tooth was then engaged mesially into the space previously occupied by the crown and after several trials was liberated out of the bone. With the employment of this technique, which consumed about thirty minutes, none of the patient's teeth were traumatized.

Examination of the bone overlying the central and lateral roots disclosed neither evidence of their exposure nor any involvement of the bone structure itself. After the field of operation was débrided, the flap was brought back into position and retained with several sutures. Except for postoperative edema, which is usually observed after an invasion of loose cellular tissues, the patient was quite comfortable and made an uneventful recovery. Six weeks after the operation, all of the teeth were again tested for vitality, examined for firmness and color, and were found normal. The patient uses her anterior teeth in mastication and is completely comfortable in every other respect.

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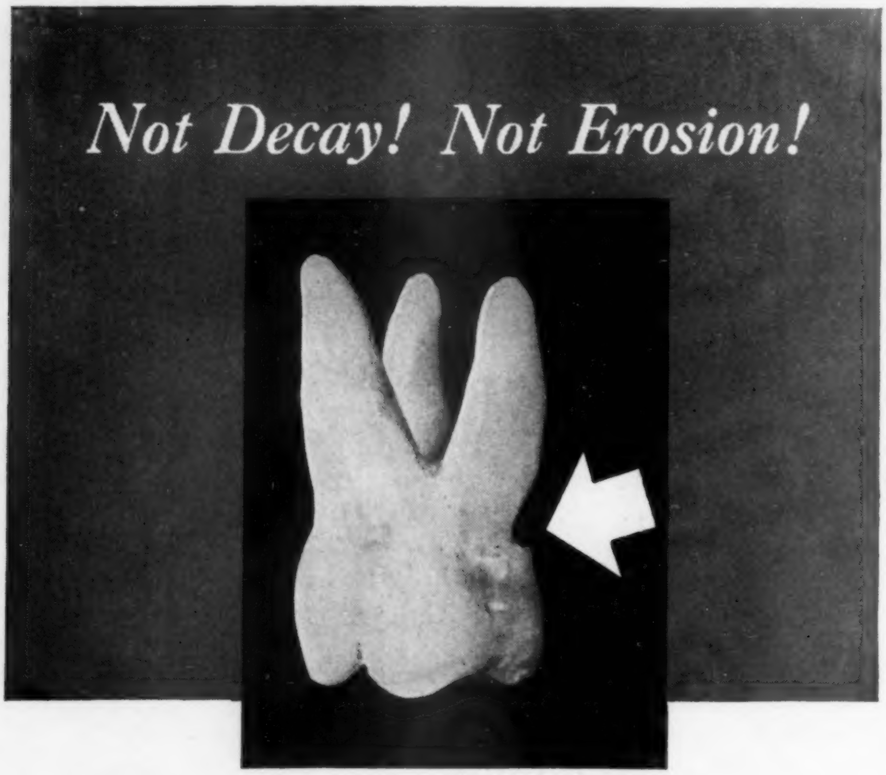
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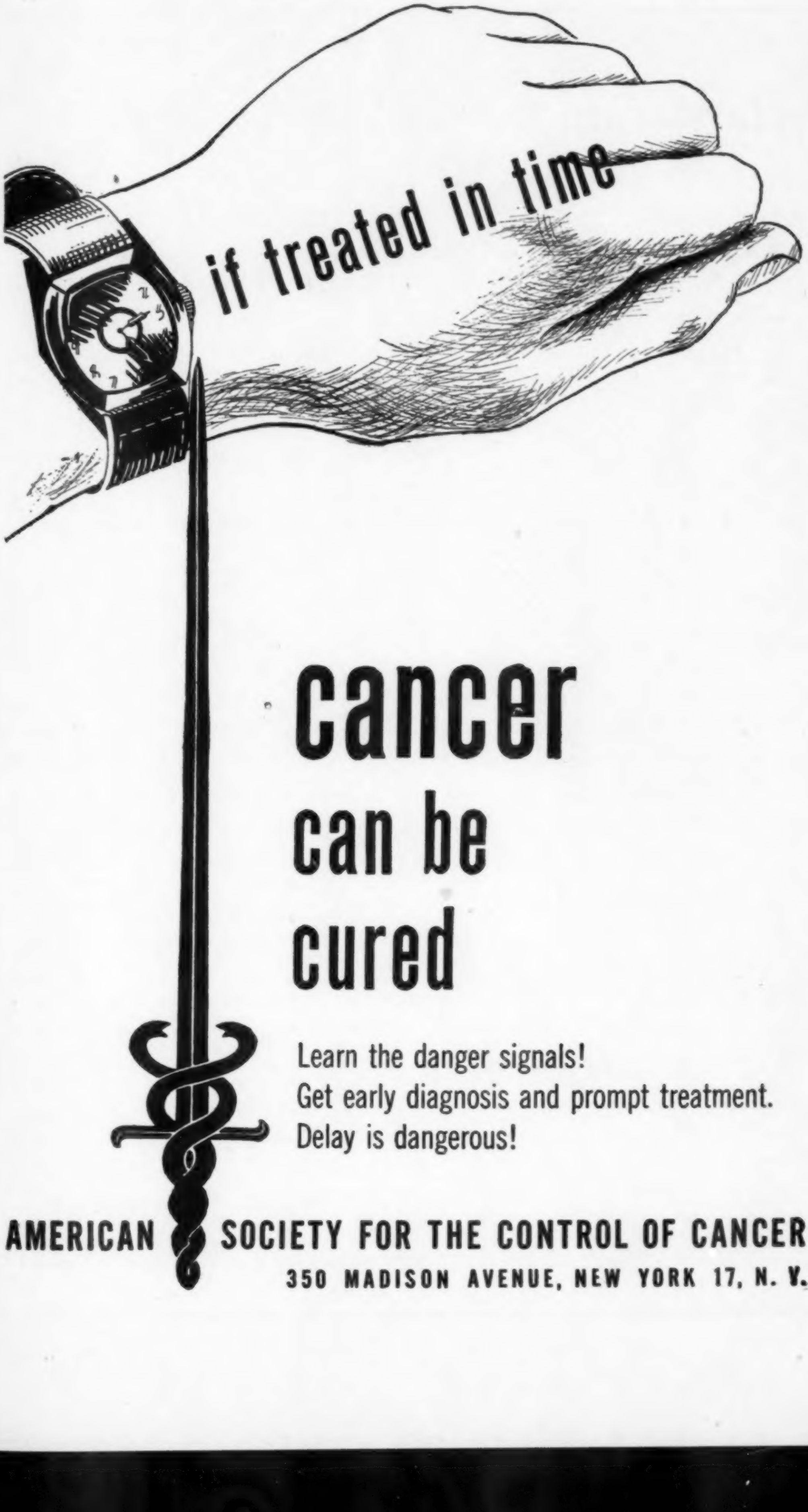
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ONE of the most difficult tasks in library reconstruction after the first World War was that of completing foreign institutional sets of American scholarly, scientific, and technical periodicals. The attempt to avoid a duplication of that situation is now the concern of the Committee.

MANY sets of journals will be broken by the financial inability of the institutions to renew subscriptions. As far as possible they will be completed from a stock of periodicals being purchased by the Committee. Many more will have been broken through mail difficulties and loss of shipments, while still other sets will have disappeared in the destruction of libraries. The size of the eventual demand is impossible to estimate, but requests received by the Committee already give evidence that it will be enormous.

WITH an imminent paper shortage attempts are being made to collect old periodicals for pulp. Fearing this possible reduction in the already limited supply of scholarly and scientific journals, the Committee hopes to enlist the cooperation of subscribers to this journal in preventing the sacrifice of this type of material to the pulp demand. It is scarcely necessary to mention the appreciation of foreign institutions and scholars for this activity.

QUESTIONS concerning the project or concerning the value of particular periodicals to the project should be directed to *Dorothy J. Comins, Executive Assistant to the Committee on Aid to Libraries in War Areas, Library of Congress Annex, Study 251, Washington 25, D. C.*


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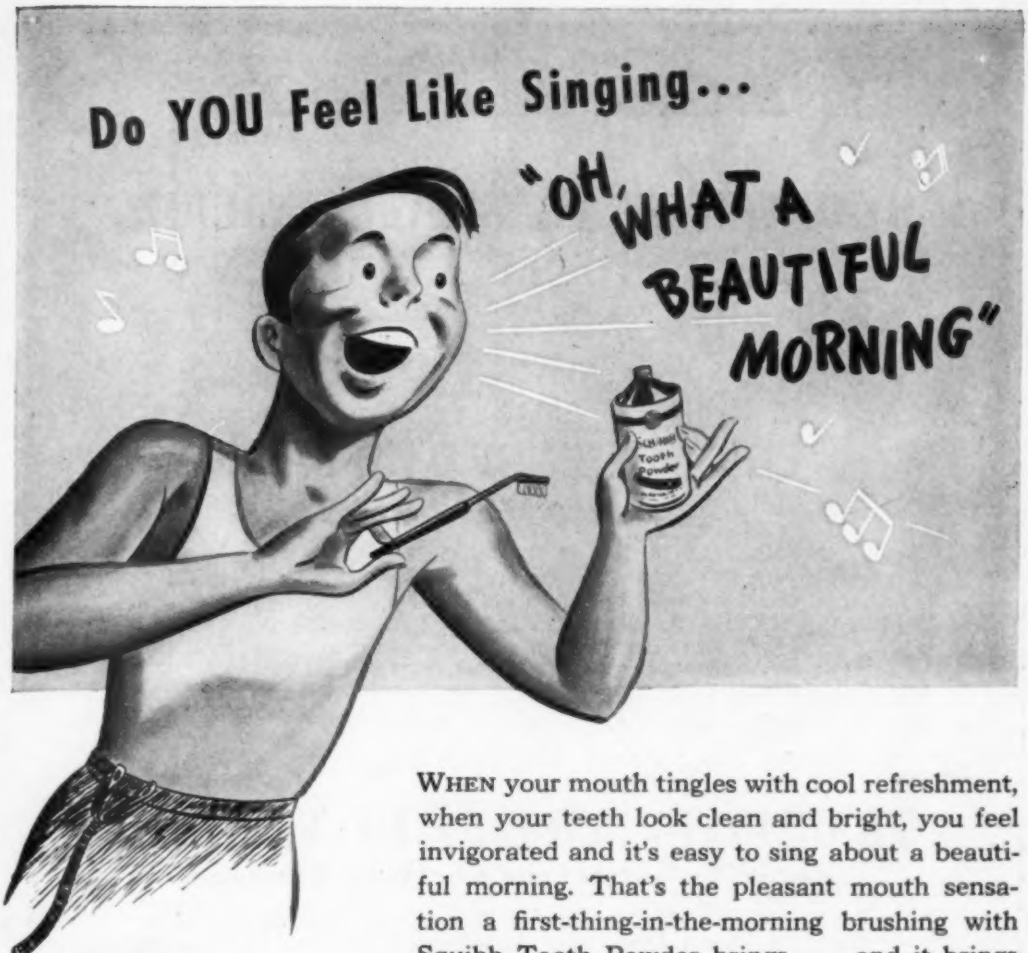

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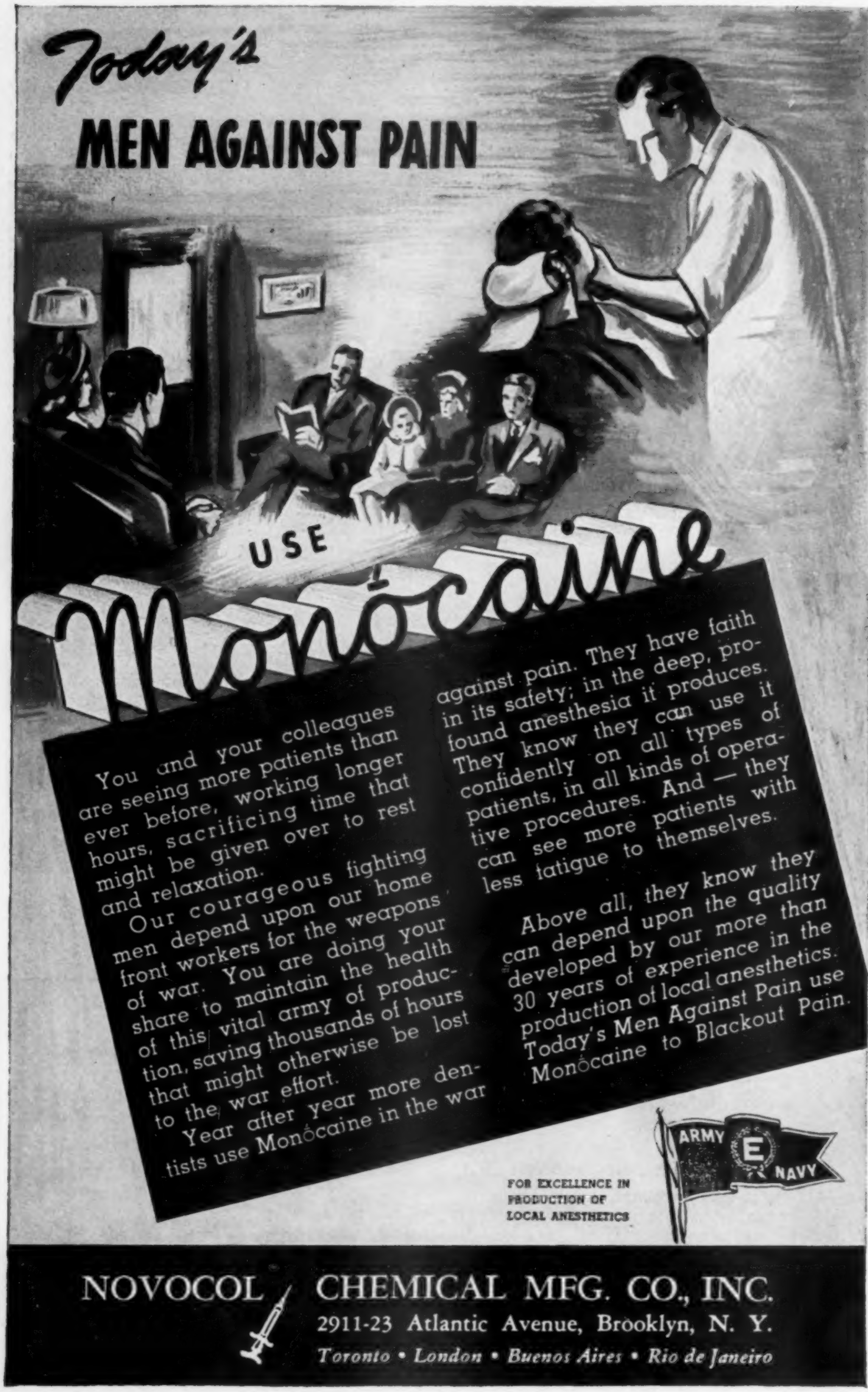

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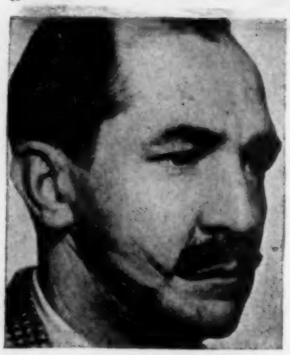
II 217 DAYS



III 566 DAYS

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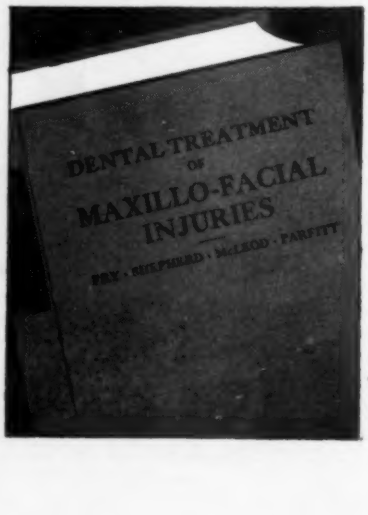

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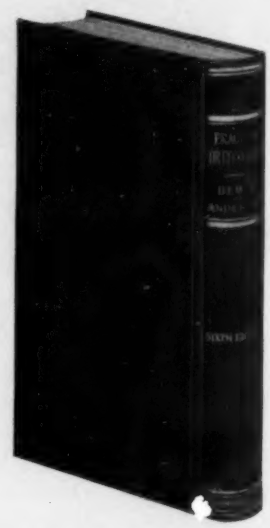

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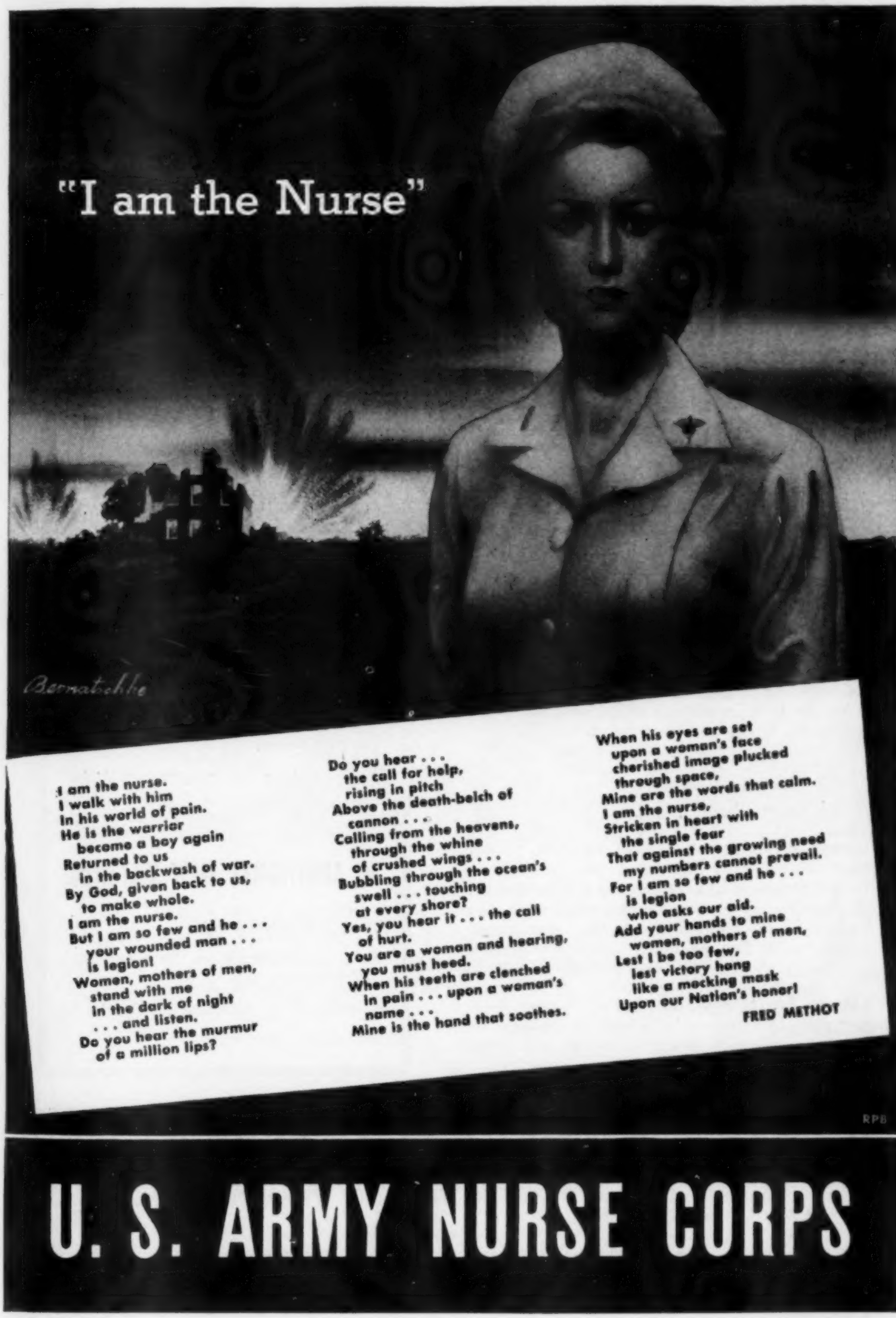
"I am the Nurse"
I am the nurse.
I walk with him
In his world of pain.
He is the warrior
become a boy again
Returned to us
in the backwash of war.
By God, given back to us,
to make whole.
I am the nurse.
But I am so few and he . . .
your wounded man . . .
is legion!
Women, mothers of men,
stand with me
in the dark of night
. . . and listen.
Do you hear the murmur
of a million lips?
Do you hear . . .
the call for help,
rising in pitch
Above the death-belch of
cannon . . .
Calling from the heavens,
through the whine
of crushed wings . . .
Bubbling through the ocean's
swell . . . touching
at every shore?
Yes, you hear it . . . the call
of hurt.
You are a woman and hearing,
you must heed.
When his teeth are clenched
in pain . . . upon a woman's
name . . .
Mine is the hand that soothes.
When his eyes are set
upon a woman's face
cherished image plucked
through space,
Mine are the words that calm.
I am the nurse,
Stricken in heart with
the single fear
That against the growing need
my numbers cannot prevail.
For I am so few and he . . .
is legion
who asks our aid.
Add your hands to mine
women, mothers of men,
Lest I be too few,
lest victory hang
like a mocking mask
Upon our Nation's honor!
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